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(DEPARTMENT OF VETERINARY SCIENCE)

Swamp Fever in Horses

By L. Van Es, E. D. Harris
and A. F. Schalk

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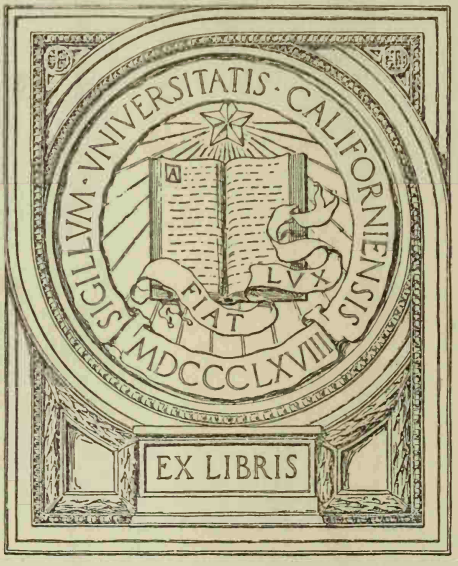

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Swamp Fever in Horses

By L. Van Es, E. D. Harris and A. F. Schalk

(equine infectious anemia)

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SWAMP FEVER IN HORSES

(From Department of Veterinary Science.)

By L. VAN ES, E. D. HARRIS and A. F. SCHALK

For some years past a peculiar disease of horses, widely known under the name of swamp-fever, has engaged the attention of veterinarians and horse owners of various sections of the Northwest. Also in our own state, the disease has occasioned considerable damage and in order to obtain some definite knowledge on the subject, this department has been entrusted with a systematic investigation of the disease. Other institutions and persons also have been engaged in research work on the disease and it may not be amiss to sum up the results of the investigations as well as the more important features of the disease as we meet it in the field.

It is uncertain when and where the disease first began to attract attention. According to Torrance [12], veterinarians settling in the Province of Manitoba during the years 1881-1882 soon had their attention called to it and to the fact that the disease was at first confined to the country bordering the Red River. From here it seems to have spread over a large area of Manitoba and into some parts of the Northwest territories.

A disease, of which it is fair to suppose that it is identical with "swamp-fever" is described by Watson [6], who writing in 1896, states that the disorder first made its appearance in the vicinity of Eau Claire in the State of Wisconsin some seven or eight years before. Since then it had continued its distribution covering an area with a radius of about one hundred miles.

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In 1899 Taylor [7] describes the Manitoba disease, but it was not until 1902 that a full scientific description of the disease was offered by Torrance [9].

In addition to the published reports of Torrance [9-12-15-21-28-54], Rutherford [11], Ballah [31], Charlton [33], Acres [48], McGilvray [64], and Todd and Wolbach [75] contributed to the Canadian literature on the subject.

From Minnesota the disease is mentioned by Brimhall, Wesbrook and Bracken [10] in 1903 and by Beebe [18] in 1905.

From Nebraska, the disease is described by Peters [26] in 1906; from North Dakota by Van Es [37,68] in 1907 and 1910; from Texas by Francis and Marsteller [42] in 1908; from Nevada by Mack [50-51-65-73] in 1909 and 1910; from Louisiana by Flower [57] in 1909; from Wyoming and Colorado by Whitehouse [56] in 1909; from Oklahoma, Kansas and Missouri by Kinsley [63-72] in 1910; from Washington by Thatcher [71] in 1910 and from the delta country of the Mississippi and Arkansas by Norton [82] in 1911.

General descriptions of the disease were published by Mohler [46-52-53-66] in 1908, 1909, and 1910.

Darling [58] writing from the Panama Canal Zone, also makes mention of the disease clinically corresponding to the swamp-fever of the United States.

While it is by no means certain that there is always described under the name of "swamp-fever" one and the same disease, there is sufficient evidence to show that we have among the horses of a large area of the continent, a disease, which has thus far only received purely local attention.

On the European continent also there seems to exist a disease, which is exceedingly similar to ours (at least for so far as that of the Red River Valley is concerned) and probably identical to it.

The disease has been described by various French writers since 1843. In more recent times something like our swamp-fever was observed in 1883 by Zschokke [1] in Switzerland; in 1886 by Fröhner [2] in southern Germany;

in 1890 by Ostertag [3] in Berlin; in 1901 by Köpke [8] in the vicinity of Metz; in 1904 by Jarmatz [14] among the farm horses of Lorraine and by Vallée and Carré [16] in various sections of France; in 1906 by Béghin [22] near Florennes in Belgium; in 1906 and 1908 by Ries [27-47] in Luxemburg; in 1907 by Brickman [32] in Sweden and by Charon [34] near Sainte Menehoulde; in 1907 by Ostertag [35] in the vicinity of Trier; in 1908 by Friedrich [44] in Kur-Hessen; and, in 1909 by Hutyra and Marek [69] in Hungary.

It is not quite certain that the American disease is identical to the one described, but there are very strong indications that it is.

The name under which the disease is most widely known in this country plainly indicates that it is most prevalent in low, marshy sections or during wet seasons. Torrance [9] states that the worst infected districts are notably those nearest to sea-level and during wet seasons the losses there are very great. In a later report [15] he points out that the ebb of the disease in Manitoba, which had a dry summer, appears to coincide with an increase in the Northwest territories, where the season was unusually wet, a fact which lends support to the theory of swamp-infection as the main source of the disease.

In the investigation of the disease in Minnesota by Brimhall, Wesbrook and Bracken [10], two distinct foci of distribution came under observation. In one focus, the same geologic formation generally prevails. The soil is a heavy black loam, while the country is markedly flat and there is but little natural drainage. Bogs and small lakes and ponds are very apt to form, wherever flowing wells are present. Wherever the land is a little lower than the surrounding country, water will stand on it for a considerable time each year or there may be an abundant growth of swamp-grass. The second focus of the disease did not show the same uniformity in geologic features and topography,

nor was the disease as generally prevalent as was the case in the other focus. The country is rolling, with an exceptionally light sandy soil, in many places well wooded. In neither of the two districts could low places be excluded, although the observers failed to find conclusive evidence of the relation between those circumstances and the occurrence of swamp-fever.

Rutherford [11] states that the disease is most prevalent in a low lying swampy country. Peters [26] found the disease on marshy pastures during wet seasons. Francis and Marsteller [42] describe the disease as occuring in the flat coast region of their state. Acres [48] found the disease only on low-lying and swampy pastures where the water becomes stagnant during the hot months of the year. Mohler [52-66] finds that the disease is most prevalent in low-lying and badly drained sections of the country and considers the drainage of infected pastures indicated, as a preventative measure. According to him the disease has been found in altitudes as high as 7,500 feet on marshy pastures during wet seasons. Kinsley [63] observed that the wet seasons seemed to increase the prevalence of the disease, which, however, is as liable to be found on high land as on low.

In the description by European observers, the relations of the disease with low, marshy pastures is not emphasized, although Brickman [32] describes it as most frequent in wet regions. Ries [47] states the farms situated in wooded regions have especially been affected.

For so far as our own observations go, it would seem that most of our cases come from the lower sections of the country, although many reports indicate that the disease may occur on high and dry land, where deficient drainage could not possibly be an etiologic factor.

Most of the cases of swamp-fever occur during the months of summer and early fall. Torrance [9] finds that

the cases usually make their appearance in the month of June and increase in frequency until September and October, while Brimhall, Wesbrook and Bracken [10] state that they were not prepared to make a definite statement in relation to the time of the year when infection takes place, but that it appeared that the cases seemed to begin in the early summer months and to increase in number during the months of July, and August. Rutherford [11] speaking of the disease in Canada, states that it is the most common in the late summer and early autumn, but that a few fresh cases come under observation between December and the latter part of July. In the outbreak described by Mack [50] the disease appeared during the summer of 1906. It abated during cold weather but only to reappear with equal fatality during the following summer, but a year hence there were but few sporadic cases. Mohler [52] found that the disease makes its appearance in June and increases in frequency until October. The observations of Kinsley [63] show that swamp-fever is most prevalent in the summer months, the initial attack usually occurring during July, August or September.

Among the European data on the disease, we find that the outbreak described by Köpke [8] also occurred during the latter part of the summer and autumn, while Ries [47] confirms an estivo-autumn recrudescence of the malady observed before.

The foregoing observations are fully in accord with the usual occurrence of swamp-fever as it is met with in North Dakota. The great majority of our cases show up for the first time during the months of August and September, and it is probable that cases which occur during the winter contracted the disease during the preceding summer or autumn.

It is difficult to undertake to place an estimate on the economic importance of the disease. While we cannot express losses in money values, there seems to be no question as to the serious nature the disease may assume. Ruther-

ford [11] reports that in some of the Canadian districts the mortality is simply appalling and that settlers were ruined by the loss of their horses. Todd and Wolbach [75] writing about the Canadian disease state that one farmer lost fourteen horses in four years and that another near Winnipeg lost forty in the same length of time. A firm of contractors working on a railroad near Edmonton in 1908 had seventeen losses from swamp-fever in a herd of twenty-eight horses. They also quote a statement made by the animal insurance companies to the effect that seventy per cent of the death claims for horses made in Manitoba are on account of swamp-fever. In other parts of the country, the disease is entirely unknown, while in a few sections sporadic cases occur from time to time. In one of the Red River Valley counties of North Dakota, where the disease has been prevalent for 8 to 10 years, thirteen farms with a total horse population of 242 head sustained a loss of 98 head or about 40.5 per cent during the year 1908. On another farm in the same district, twenty-four horses succombed to the disease within four years and the loss forced the owner to close out his business. Numerous instances of this sort are recorded for the different "swamp-fever" districts, and fairly indicate the havoc of which the disease is capable. Those figures also point to the serious possibilities in case the disease should become more generally distributed. We should not pass by "swamp-fever" figures, however, without a word of caution against their acceptance at their face value.

It is probable that only those cases of the disease which go on to the chronic anemic stage are diagnosed as such and that the more acute forms of it are never properly identified. On the other hand, there is a strong tendency on the part of veterinarians and horse owners, to regard any chronic disease accompanied by anemia as swamp-fever, while in not a few instances anything more or less difficult in diagnosis or in which a diagnosis at the spur of the moment is impossible is being charged to the disease under consideration.

In the light of our present knowledge of the disease, swamp-fever may be defined as a specific septicemia of the horse species, due to the presence and vital activity of virus, which thus far has been demonstrated only in an ultramicroscopic form and characterized by a more or less intermittent fever and albuminuria, accompanied or not accompanied by a progressive anemia.

Various views have been advanced in regard to the etiology of "swamp-fever." Zschokke [1] writing about an anemic disease of horses, as seen in Switzerland, was led to think that the disease was caused by an agent similar to that of malaria. Fröhner [2] also points to the probability of some infection as an etiologic factor, and in support of such a possibility, he mentions the simultaneous sickening of several animals in one stable, the sudden febrile attacks in the beginning and the malaria-like exacerbations and remissions of the fever during the further course of the disease.

On the other hand Watson [6] expresses himself, that there is no doubt but that it is caused by a vegetable poison taken into the system through the mouth. Köpke [8] believes that the disease described by him is one of infection, although he was led to doubt, because it happened frequently that horses exposed to infection did not become sick at all.

In the first description by Torrance [9] he states that the question of contagion was not settled. He had kept fever cases in the most intimate contact with healthy horses without observing transmission of the disease, while his inoculation experiments had up to the time of writing proved negative. He also states that there appeared to be reasons to think, that paludism plays an etiologic part, basing this opinion upon the prevalence of the disease in low-lying districts. In another article, Torrance [12] expresses himself as certain that the disease is not identical with surra and mentions a large bacillus, non-motile, spore-forming and

Gram staining as having a possible connection with the disease. Later, the same author [21] "thought" that he saw several round bodies in the erythrocytes which occurred either singly or arranged in groups of four. Still later Torrance [28] reports that in his investigation a point was reached at which it seemd reasonable to exclude trypanosoma or plasmodia from the possible causes of the disease. In a publication of still later date, Torrance [54] reports a case in support of the view that a disease closely simulating swamp-fever in its clinical manifestations, may be accompanied or caused by the presence of large numbers of worms belonging to two closely allied species (Sclerostomum tetracanthum et armatum). The suggestion of a hemolytic agent, that may originate in the intestinal tract by bacterial activity is made by Torrance [21].

In the investigations in Minnesota by Brimhall, Wesbrook, and Bracken [10], the possibility of horses of the infected districts drinking from pools, marshes or lakes could not be excluded, while attention is called to the possible part played by insects or arachnoids as factors in "aiding saprophytic development of parasitic bacteria," as well as to the overworked condition of horses and their irregular food supply as influencing their resistance power to infection. The same investigators made very exhaustive bacteriologic studies with the material at their disposal and report that a small, non-motile, ovoid bacillus, which they named B. equisepticus, was present in practically all cases. It was further found that B. equisepticus was highly virulent to rabbits, pigeons, sparrows, while calves were less susceptible. Dogs and swine appeared to be resistant. Horses succumbed to the infection and "at autopsy, the findings were practically those met with in clinical cases of acute swamp-fever."

Other experiments were also carried on with an organism spoken of as B. pyrogenes equinus as well as with the toxines of both organisms mentioned. The authors mentioned, further state that an etiologic relationship between

B. equisepticus and the disease "swamp-fever" would therefore seem to be a safe deduction from the autopsy findings in clinical cases and the experimental inoculations.

Jarmatz [14] is of the opinion that the disease begins early in life, during the first months even and attributes it especially to the results of in-breeding, which is very much in vogue in the section of Lorraine, where he made his observations. Insufficient nutrition also shares in the production of the disease.

Bacteria were isolated from most cases by Beebe [18] but his experiments did not seem to establish a causative relationship of those micro-organisms to the disease. In the same report, the author mentions the case of a horse contracting the disease after having been driven with a probable case of "swamp-fever."

Peters [26] made cultures from spleen, blood, kidneys, and urine, but those cultures inoculated into smaller animals failed to infect. His autopsies showed a large number of worms "known as tetracanthum" and he seems to lean to the theory that intestinal parasites may be etiologic factors.

Ries [27] deems the theory that intestinal helminths exercise a preponderating influence to be quite supportable and calls attention to the fact that he once made the statement that he accused the larvæ of Gastrophilus to play a part in the propagation of pernicious anemia, either by being the carrier of infection, by opening up a port of entrance or by causing a general weakening of the resistance power. Ries thinks that the frequent occurrence of the disease on isolated farms situated in wooded regions supports his theory.

Ballah [31] injected some cavia with "swamp-fever" blood and the animals promptly became sick. It was proven, however, that those results could be attributed to a streptococcus, which occurred in the blood of a horse as a secondary infection. This author was induced by certain changes found in the liver to study this organ more closely for the

presence of micro-organisms, and in the course of this investigation he found certain liver-cell inclusions, which he believed to the protozoa and probably the specific cause of the disease. Those bodies were found by Ballah in over 50 per cent of the cases examined, but they were absent in all specimens of normal liver from horses or other animals.

Charlton [33] injected several laboratory animals with material from swamp-fever cases. After two weeks, some of the cats showed a slight rise in temperature and appeared somewhat ill. The fever was irregular and although the animals ate well, they failed to thrive and had an unhealthy appearance. One of the cats developed a well defined edema all along the belly about five weeks after injection. The edema lasted about two months, when it disappeared, the animal afterward remaining in good health. Two of the smaller cats died about three months after injection, but horses injected with the blood of the sick cats remained in good health. Specific organisms were not found.

Brickman [32] in cases, in many respects similar to the pernicious form of anemia in France, claims to have found parasites resembling those of malaria in the blood corpuscles.

Van Es [37] was able to isolate several bacterial species from the blood of "swamp-fever" cases and his observations on the damage done by certain helminths may be an indication as to how the micro-organisms found their way into the general circulation. There was no evidence adduced, however, that the conditions found bore an etiologic relation to the disease under investigation.

Hundreds of blood examinations were made by Francis and Marsteller [42] but none of them revealed the presence of blood parasites. Cultures from the blood on various media also led to negative results. In a later report the same authors [81] described an experiment on the possibility of transmission of infectious anemia through the agency of

Fig. 1. A field case, No. 921, about one month before death.

Fig. 2. The same horse as shown in Fig. 1 about one week before death. Note the marked edematous swellings of brisket, lower part of chest and abdomen, the sheath and the right hind leg.

Böophilus annulatus, but in the one case tried the results were negative.

Mack [50] isolated various bacteria but their relationship to the disease under consideration was not positively demonstrated, neither did he find evidence of direct contagion.

The invariable presence of parasitic aneurism in all the cases subjected to autopsy by Whitehouse [56] led him to look with considerable suspicion upon this fact.

Trypanosoma were found by Darling [58] in the blood of an American gelding and a number of mules in the Panama Canal Zone. These animals were suffering from a disease which corresponds clinically with the descriptions of "swamp-fever" published in this country. The readiness with which Darling infected a great assortment of various animal species with his trypanosoma renders the identity of the Panama disease with our swamp-fever rather questionable, however. His inoculations with filtered blood did not permit of definite conclusions and hence the reports of further experiments by this investigator will be looked forward to with considerable interest.

Kinsley [63] reports the instance of a farm, which has been infected for twelve or fourteen years and on which horses were lost with the disease every year, while on a neighboring farm not more than sixty rods away, there never was a case. On another farm five horses were lost during the winter and a pony placed in the same stable for three or four weeks during the following summer became affected and died. On the other hand, several instances were observed in which one of the horses of a certain team had become infected and the other remained normal, and this, in spite of the fact, that the animals were watered from the same pail, and fed in the same trough. Colts appear to suck from infected mares with impunity, while in other instances sucking colts become affected, although the mares remain healthy. Horses and mules seem equally susceptible, while the age of the animals seems to have no

more influence on susceptibility than the breeds to which the animals belong.

It is probable that Torrance [12] was the first to actually transmit the disease by direct infection, although from his report it does not appear that he clearly recognized the fact. In 1902 this investigator observed a rise of temperature in a horse injected with swamp-fever blood on the 12th day after injection and he merely holds it for probable that the animal had become infected. This horse had two more subsequent rises of temperature at intervals of 10 to 12 days, but without any clinical symptoms of the disease.

The first serious attempt to clear up the etiology of the disease was made by Vallée and Carré [16-17-19-23-36] and they succeeded in doing so to a remarkable extent, at least, for so far as the European disease was concerned. While the infectious nature of the disease had been suspected for some time, they definitely settled the question of transmitting the disease to a healthy animal by the injection of blood from a diseased one. In their first communication Vallée and Carré [16] relate that in their first experimental subject, the disease ran its course, identical to the natural disease in 57 days and how the blood count dropped from the normal 7,800,000 to 2,800,000 on the day of death. The autopsy revealed the ordinary lesions of anemia, namely extreme muscular emaciation, subcutaneous, sub-serous, and peri-ganglionic edema, enlargement of the spleen, liver cirrhosis and hemorrhages of the bone-marrow. Bacteriologic research was not rewarded with positive results and a search for piroplasma or trypanosoma also failed to disclose those parasites. On the supposition that the etiologic factor of the disease might belong to the invisible viruses, Vallée and Carré made a mixture of 500 c. c. of blood serum coming from a sick animal with 200 c. c. of physiologic salt solution and added to it a culture of an extremely virulent ovine pasteurella. This mixture was passed through a special filter bougie and the opalescent liquid passing through the filter was injected in doses of 20 c. c. into the veins of some

rabbits and into the peritoneum of cavia, which animals remained healthy. This experiment showed the efficiency of the filter. Next the investigators injected 500 c. c. of the filtered liquid into a healthy horse and found that after a lapse of a certain period of incubation the animal presented symptoms of an absolutely typic anemia. It was thus fairly well established that the etiologic factor in the disease is a so-called ultra-microscopic organism.

Somewhat later Vallée and Carré [17] completed their previous report and state that it is easy to maintain the virus by successive passages through the horse, the virulence increasing. They also found that the anemia in reality only constitutes one of the forms of an infectious disease, which often reveals itself under an entirely different aspect. In fact, three types of the disease may be recognized and Vallée and Carré emphasize that those three types unquestionably belong to one and the same affection as they are experimentally reversible. They also infected the ass and showed that virulent blood loses nothing of its virulence by being diluted five times and by being filtered either through a Berkefeld filter or through Chamberland bougies F or B. In the same series of experiments, Vallée and Carré also proved that the disease is transmissible by the digestive tract, as a horse fed on 20 c. c. of virulent blood promptly contracted the disease. Note is also made of the fact that horses which were apparently absolutely cured of the chronic form of the disease still retained their full infective powers and hence Vallée and Carré express the opinion that the presence of such virus-carriers constitutes in infected regions a great obstacle to the extermination of the disease by prophylactic measures.

Carré and Vallée [19] in a later report cite an instance which tends to show the infectiveness of the urine of an apparently recovered case. Continuing their studies Carré and Vallée [23] found that the quantity of virulent blood injected into a healthy experimental animal had no influence upon the course of the disease.

Cattle, sheep, goats, dogs, rabbits, cavias, mice and white rats proved to be refractive to infection.

Carré and Vallée report that the virus is destroyed by heating to 58° C. for one hour, but that drying in vacuum at room temperature does not alter its virulence. The dried material from 1 c. c. of virulent blood kept for ten days and then inoculated into a vigorous horse, kills the same in thirty days, but after storing this substance for seven months, it is no longer disease producing.

The authors metioned, further report that the keeping of virus outside of the body seems to rob it gradually of its virulence, but that putrefaction did not seem to disturb the vitality of the virus.

Carré and Vallée are of the opinion that the natural mode of transmission of the disease takes place by food stuffs and drinking water soiled by virulent urine and probably also the fæces, but they did not succeed in establishing any relationship between blood sucking animals and the distribution of the disease.

The results of the experiments of Vallée and Carré relating to the ultra-microscopic nature of the virus have since been confirmed by various investigators, Charon [34], Ostertag [35], Francis and Marsteller [42-81], Hempel [49], Mack [50], Mohler [52-66], Van Es [68]; Todd and Wolbach [75] transmitted the disease by intra-peritoneal injection.

In the experiments of Ostertag [35], attempts to transmit the disease by means of the saliva failed, but he succeeded to bring about infection with 20 c. c. of blood serum of an acute case, after it had been kept on ice for seven weeks.

Hempel [49] reports that virulent material which was stored on ice for some time and given by the mouth in small quantities was not capable of producing the slightest reaction, although it produced, a typical infection when given subcutaneously in one-fourth of the dose used in the feeding experiments. Material from a case in the dormant stage of the disease, when given intravenously in doses of

120 c. c. produced a slight form of the disease and when given per os in a dose 2½ times as great, a one day fever period was observed after 21 days incubation. Given intravenously or subcutaneously, small amounts of virulent blood will produce infection, but larger quantities are required if infection per os is to be produced.

The question of a transmission of the disease by contagion has not received much attention, but the few data on record seem to indicate that this means of transmission is not an easy one.

Melvin [70] reports the experimental exposure of a healthy horse, which was quartered in a stall adjacent to one containing a sick horse for seven months and which failed to transmit the disease.

Francis and Marsteller [81] kept a susceptible pony continually exposed to swamp-fever infection for more than two years by intimately associating it with infected animals in a small pasture, without the experimental pony becoming infected.

In our own experiments with the disease as it occurs in the Red River Valley, the results obtained by Vallée and Carré were practically repeated.

During the latter part of 1907, we injected an apparently healthy horse intravenously with 8 c. c. of blood from a typical chronic field case and the injection was followed some eight days later by the initial fever attack, which we have since learned to recognize as evidence of positive infection. Since that time, we have succeeded in transmitting the disease by means of filtered (sterile as far as cultures are concerned) diluted serum.

In our hands, not only the disease was transmitted by subcutaneous or intravenous injections or virulent blood, but we also produced infection by giving capsules filled with virulent blood by the mouth. In addition, we found that urine either injected subcutaneously or given by the mouth is capable of transmitting the virus.

For so far as our investigations go, it does not appear that the virus is eliminated by the bowels. The subcutaneous injection of 100 c. c. of an extract of fæces from a virulent horse into a susceptible horse was not followed by any reaction. In another case four liters of a similar extract in which 1790 G. of fæces from a virulent case were used and which were introduced into the stomach by means of a stomach tube failed to bring forth any noticeable results.

The latter experiment was repeated with larger quantities of extract made with fæces from another case, using a different experimental horse, with the same results. The susceptibility of the experimental horses used was proven by their subsequent infection when injected with virulent blood.

From the various experiments quoted, it seems safe to conclude that the disease is due to an ultra-microscopic virus and that this is the primary factor to be considered in the problem. The experiments have eliminated poisonous plants as a factor, and the readiness by which a virus can be caused to make several passages in unbroken succession also sets aside the etiologic importance of intoxication by intestinal helminths. There can be no question as to the possibility of intestinal worms in producing outbreaks of anemic disease, and in the study of such diseases, their presence should not be neglected, but in the disease under consideration, they cannot be regarded as primary factors.

The importance of certain helminths as producers of anemia and as instruments of opening the way for bacterial invasion is strongly pointed out by the publications of Glage [24] and of Weinberg [38-39].

For the time being, the demonstration of ultra-microscopic virus must not be accepted as final, and the search for blood micro-parasites should not be discontinued. We may readily conceive the possibility that in the metamorphosis of a micro-parasite there may occur a stage, during which the forms become filter passers.

The nature of the virus was not further investigated by the writers, with the exception of its behavior toward freezing temperatures.

The following observations were made:

A quantity of virulent blood was exposed to freezing for 24 hours during which the minimum temperatures recorded was 10° F. The blood was permitted to thaw at room temperature and 240 c. c. were injected subcutaneously into an experimental horse with the result that the animal sickened on the ninth day and died on the fifteenth day after injection.

Another quantity of blood was exposed to freezing, the prevailing temperatures being indicated by the following table:

Date	Maximum Temp. F.	Minimum Temp. F.	Date	Maximum Temp. F.	Minimum Temp. F.
Jan. 3	-10	-26	Jan. 20	17	0
Jan. 4	16	-22	Jan. 21	14	-15
Jan. 5	0	-14	Jan. 22	3	-13
Jan. 6	3	- 7	Jan. 23	2	-12
Jan. 7	15	-14	Jan. 24	24	0
Jan. 8	15	-13	Jan. 25	28	- 1
Jan. 9	9	- 9	Jan. 26	27	11
Jan. 10	31	-14	Jan. 27	11	3
Jan. 11	-15	-23	Jan. 28	5	13
Jan. 12	0	-23	Jan. 29	33	3
Jan. 13	0	-22	Jan. 30	16	-13
Jan. 14	-11	-22	Jan. 31	23	- 9
Jan. 15	- 2	-19	Feb. 1	24	7
Jan. 16	- 3	-20	Feb. 2	23	-16
Jan. 17	8	-10	Feb. 3	10	-16
Jan. 18	8	- 7	Feb. 4	11	-14
Jan. 19	22	0			

On the fifteenth day of exposure, a quantity of this blood was thawed and 240 c. c. of it injected into an experimental horse. This horse developed fever after an incubation period of forty-nine days and died eight days later.

A similar quantity of this blood was injected into another experimental case after it had been exposed for thirty days with the result that the animal developed fever on the 45th day, dying 6 days later.

From the above observation it would seem that if severe freezing has any effect at all on the virulence of the blood, it merely consists of lengthening the incubation period. Before concluding definitely on this point, however, further research will be required, especially in view of the unusually long period of incubation.

In certain cases of pernicious anemia of man, the presence of peculiar bodies were found by Perles (5). *He proposes the name of anemia-bodies for them and describes them as elongated, elliptical, very thin and narrow, flexible, colorless, and highly refractive leaflets. Perles failed to either stain or cultivate them, nor could he find them in cases of secondary anemia. Hoefer* (62) *apparently found certain protozoa in a case of severe anemia eight days after the beginning of sickness, but the question whether or not they constituted an etiologic factor could not be decided.*

The transmission of "swamp-fever" to other animals seems to be denied by most authors. Carré and Vallée (23) tried to infect other animals, but without positive results. Ostertag (35) reports that in the district where he studied the disease, a transmission to other domestic animals had never been observed and attempts to transmit the disease artifically also resulted negatively. Man had not been known to contract the disease, although many persons constantly cared for infected horses and even consumed their meat. Francis and Marsteller (42) failed to obtain positive evidence of infection after injecting cattle, sheep, goats, pigs and dogs with virulent blood. Charlton (33) observed that injection with virulent "swamp-fever" blood into cats was followed by illness, but the blood of the cats so affected proved to be inocuous to horses. Mack (73) in his latest publication states that in the Nevada disease there is no evidence that it is

contagious and that while there is every clinical indication, that it is a specific infectious disease, he must have more conclusive evidence than his experiments have yet afforded before one is warranted in pronouncing it such. At the time, however, he would venture no opinion as to the identity or non-identity of the Nevada disease with the affections reported from other sections. Todd and Wolbach [75] injected with swamp-fever virus four cavias, two mice, nine rats, two rabbits, ten dogs, two kittens and one sheep and no symptoms which could be attributed to swamp-fever followed in any of the animals experimented with.

In our own work, a similar condition was experienced. A number of cavias were injected with virulent blood and several among them showed a rise of temperature after a few days, while still later some of the animals died. The blood of the sick pigs injected into a healthy horse, however, absolutely failed to cause any reaction.

The occurrence of anemic diseases among live stock other than the horse is generally well known, but as far as we know, nothing like swamp-fever has ever been observed, unless the case reported by Trincas [29] in the dog pertains to a similar disease. In this case the author demonstrated that anemia in the dog may be due to an ultra-microscopic virus.

The anatomic changes met with in swamp-fever are rarely of a striking character. They include degenerative changes in the various parenchyma, petechiæ, ecchymoses, especially about the serous membranes and endocardium, edematous conditions, certain changes in the lymphnodes and the bone marrow. The changes noted indicate a more general intoxication or septicemia than that the disease stamps a peculiar feature upon a certain organ or sets of organs. In the interpretation of the various lesions found,

Fig. 3. A non-anemic field case No. 1032.

Fig. 4. Experimental horse No. 636, 33 months after original infection.

it should be borne in mind that in all probability secondary infections and influences also bear a large share in their production and that it is not always possible to determine which lesions to charge to the specific agent of "swamp-fever" and which are to be credited to other determining factors. For the purpose of presenting the pathologic anatomy of swamp-fever as fully as the importance of the subject demands, it seems wise to quote from the descriptions of various observers in addition to mentioning our own findings.

Taylor [7] found the blood of the carcass darker and more fluid that normal. Numerous petechiæ and hemorrhagic spots were visible in the fasciæ of dorsal and lumbar muscles. Both heart cavities were thickly spotted with black hemorrhagic areas. The intestinal surfaces were also studded with petechiæ. The mesenteric lymphnodes were black as if filled with dark blood. The spleen was considerably enlarged. The kidneys showed nephritis (interstitial). The muscles showed hemorrhages both in the perimysium and between the fibres themselves. Taylor states that numerous black bodies scattered throughout the lungs are "the most striking features."

In the experience of Köpke [8] the carcasses dead with the disease were highly emaciated, hide-bound, and presented a dull, rough coat. The mucosæ were perfectly anemic and the eye-balls were sunk deeply into the orbits. The subcutaneous veins appeared almost bloodless and the subcutis of the thorax, belly, prepuce, and the hind legs were markedly edematous. The musculature, which immediately after exposure, had a pale red color, assumed a characteristic reddish yellow, almost orange red tint after an exposure to the air for a few hours. The mesocolon showed numerous hemorrhages of the size of a pea to that of a hazelnut. The mesenteric lymphnodes were two or three times their normal size on account of the edematous swelling. The mucosa of the intestines showed petechiæ in two of the cases. In the third case, the mucosa was thickened.

this thickening being firm, and more of a fibrous connective tissue nature than edematous. Liver and spleen were normal in size, the former showing a slight parenchymatous cloudiness. The markedly enlarged kidneys appeared pale red and were so friable that their entirety could scarcely be preserved, when they were taken out of their capsule. Their parenchyma was cloudy. The heart was always considerably enlarged and the myocardium degenerated to a high degree so that it presented a pale-grey-red color and a parboiled appearance. In the ventricles, especially about the columnæ carnæ, extensive endocardial hemorrhages could be demonstrated.

Torrance [9] describes anemaciated state of the carcass with a most remarkable absence of adipose. The tissues were blanched. The blood is often firmly clotted in all the vessels, the clot having the appearance of yellow jelly. The abdominal viscera are normal in color but frequently studded with petechiæ on the serous surface. The spleen is generally much enlarged, reaching in some cases a weight of six pounds, while its surface appearance is normal. The liver is not much changed in appearance, but is sometimes studded with flakes of lymph. The kidneys, not infrequently, show evidence of chronic nephritis. A few petechiæ may be noted on the surface of the lungs. The heart is generally enlarged, sometimes much hypertrophied, reaching in one case a weight of twelve pounds and fourteen ounces. The pericardium is frequently studded with petechiæ and the pericardial fluid in some cases is greatly increased. Spinal cord and brain are found to be in a normal condition.

Brimhall, Wesbrook, and Bracken [10] report sharply defined edemata in the regions of the chest, the abdomen and the limbs. In some cases there was evidence to show that such subcutaneous edematous areas became purulent later on. Conditions of this kind were met with between the pectoral muscles and those of the legs. They were also seen in the mediastinum and in the auriculo-ventricular

groove in advanced cases. The deeper layers of the skin and the subcutaneous connective tissue showed in nearly all cases hemorrhages, varying in size from petechiæ to irregular spots, two or three inches in diameter. Such hemorrhages were also found between and in muscles, fasciæ, synoviæ, lymphnodes, serous membranes and the heart. The lungs were sometimes stippled with red spots, showing through the pleura, while at other times irregular areas of hemorrhage occurred which completely filled several contiguous lobules. The spleen as a rule was found to be spotted with irregular red blotches and in certain instances well marked and extensive infarcts were to be seen. In a few instances the organ seemed almost entirely devoid of hemorrhages. The changes shown by the kidneys varied and included petechiæ, large hemorrhages under the capsule and parenchymatous nephritis. Hemorrhagic areas were quite frequently seen in the mesenteries and the walls of the stomach and intestines. In some cases they were several inches in diameter. Often they were sub-peritoneal, but in a number of instances they involved the entire intestinal wall. While the authors found it impossible to ascertain accurately at what stage of the disease hemorrhages are most likely to occur, it would appear that they were more common in advanced cases, when the blood changes were most marked. Abscesses were frequent in the Minnesota cases and the inflammation of the serous surfaces was so common as to be recorded as characteristic of the disease. The heart was enlarged, in many cases showing numerous hemorrhagic patches underneath the endocardium or epicardium and occasionally involving the whole thickness of the heart wall. Frequently there was an increase in the pericardial fluid and very occasionally a sero-fibrinous pericarditis was present. In some instances, white depressed areas, irregular in shape and size probably indicated the seat of old hemorrhages and infarcts. Histologic examination showed that in those areas the heart muscle fibers are replaced by connective tissue. The lymphnodes are

frequently swollen and infiltrated with a gelatinous exudate. In all cases lymphnodes could be found in which marked hemorrhages could be demonstrated and the whole structure was often found to be hemorrhagically infiltrated. The liver usually was increased in size and congested and when peritonitis was marked flakes of lymph covered its surface. The liver tissue was frequently softened, light in color, giving evidence of parenchymatous changes. Branches of the portal vein were quite frequently found to contain whitish thrombi. Cirrhosis was met with twice. The bladder often showed hemorrhages in its wall, sometimes involving the whole thickness and the areas being some inches in diameter.

In the cases studied by Carré and Vallée [23], the lesions varied according to the evolution of the disease and were especially associated with the blood, the heart, the spleen, the lymphnodes, and the bone-marrow. In addition there were changes in the kidneys, liver, intestines and edemata of various parts. The lymphnodes in acute and sub-acute cases and also in the chronic cases during an acute exacerbation will show congestion, even hemorrhages, especially those of the mesentery and the spleen. In the cases dead with exhaustion, the nodes will show a simple edematous enlargement, while at the same time they are surrounded by a yellow edematous mass. The spleen is nearly always enlarged, doubled or tripled in volume. There is no softening, on the contrary there is thickening, the organ being lumpy in the middle and seems to have gained in consistency. On section the pulp is paler than usual and the splenic bodies are hypertrophic, granular and grayish. In acute cases, the capsule is speckled by ecchymoses. The bone-marrow most generally presents extremely marked alterations in all the patients. The lesions are particularly intense in the femurs of which the marrow is transformed for a great part of its height in a veritable bloody, brick-red or blackish mush and in consistency resembles foetal marrow. The histologic examination reveals the special

characters of the latter, like it does in all the known anemic conditions. Changes of the heart are exceedingly common. In the acute type it is speckled over by multiple ecchymoses, sub-pericardial and sub-endocardial. The myocardium is marked by numerous hemorrhagic foci, triangular in shape and of several square centimeters in area. One may observe a valvular endocarditis, this being best described by comparing theirs to the aspect of an eschar due to a weak caustic. In the chronic cases the myocardium is discolored, marked by the remains of hemorrhages, pink or greyish in color and one sees occasionally a slight valvular edema and atheromatous lesions of the aorta. The liver is generally enlarged, of a washed yellow color, sometimes stippled with fine white specks or it presents the typic aspect of the liver of cardiac insufficiency. The liver tissue is more friable and can be readily torn. The kidneys are pale, discolored in the chronic types, hemorrhagic in the acute forms. The capsule can be readily stripped. Sometimes we meet with well rounded pin-head abscesses in the cortical portion and which project under the capsule of the organ. The intestines are normal in the chronic cases, but in the acute ones on the contrary they are speckled with multiple sub-serous hemorrhages. The mucosa is the seat of a more or less intense congestion, while the muscular coat may present the same features. The lesions are generally quite extensive in the large intestines, which are sometimes entirely hemorrhagic but in the small intestines the lesions are more discreet. The lungs were always normal with the exception of a few sub-serous petechiæ.

Charon [34] notes the following changes seen during the autopsy of a natural case; emaciation, absence of a subcutaneous edema, the presence of three or four liters of a lemon-colored serous fluid in the peritoneum. The lymphnodes, the superficial ones, as well as those of the viscera, are enlarged, infiltrated, juicy, showing on the cut surface numerous hemorrhagic foci. There was an absence of internal fat, the liver in normal color, but with its cap-

sule thickened. The spleen is nearly three times its normal size and weighs 2100 grammes. The kidneys have a sclerous aspect from periphery to center. The cortical portion is discolored, the medulla is pale. A section of the bones reveals the marrow to be altered in its entirity, foetal and hemorrhagic. The endocardium is a little thickened. The same author also gives an account of the autopsy of an experimental case. He states that the cadaver is poor. There is no subcutaneous edema. The muscles have a normal color and there is no exudate in the body cavities. The lymphnodes of the groin, spleen, kidneys are very much enlarged and those of the spleen show numerous small hemorrhagic foci. There is a wasting of the internal fat. The liver has a normal color and Glisson's capsule is thickened. The spleen is triple the normal size, weighing 2600 grammes. The kidneys are slightly discolored. A section of the femur shows a hemorrhagic focus 10 c. m. in width and 1 c. m. in thickness. There is foetal marrow of the consistency of vaseline. The heart presents a hemorrhagic area of the right ventricle extending for 2 m. m. into the myocardium.

The autopsies made by Francis and Marsteller [42] revealed extreme emaciation, some edematous swellings along the lower part of the body and usually some bed sores. The muscles were pale and the blood watery. Petechiæ were noted on the heart and pericardium, while some enlargement of the heart was observed. There was a marked enlargement of the spleen, the organ ranging in weight from four to eight pounds. The digestive apparatus was normal, while kidneys and liver showed no conspicuous changes. Aside from the lesions found in the blood, heart and spleen, the authors were at a loss to account for the death of the animals.

Hutyra and Marek [69] state that the anatomic changes are those of an acute or chronic septicemia and are quite variable according to the duration of the disease. The spleen of horses, which died during an attack of the disease or during an acute exacerbation is considerable en-

larged. Its capsule is tense, marked by hemorrhages, while the pulp is black red, projecting from the cut surface, mushy or even liquified. Sometimes there are found in the otherwise normal spleen, several large projecting, uneven areas, corresponding to very dark red softened foci. The slower the course of the disease, the slighter the changes in the spleen. In very protracted cases, the enlargement is often completely absent. For so far as the corresponding organs show hemorrhagic changes, the lymphnodes of the body show acute enlargement and a hemorrhagic condition. In chronic cases, the lymphnodes may not be altered or be at the most somewhat edematous. Sub-serous hemorrhages are a common finding but in the chronic cases they become inconspicuous. They are especially seen in connection with the cæcum and the colon, but in a less marked condition also in other parts of the peritoneum. Ecchymoses are always seen under ths capsule of the liver which is more or less, in some cases, even considerably, enlarged. The intestinal mucosa shows either isolated, round hemorrhages or there is a diffuse hemorrhagic condition with a bloody consistency of the intestinal contents. The heart with the exception of that in the very chronic cases, reveals sub-peri, sub-endo, or intramyocardial hemorrhages. The kidneys show hemorrhagic areas only in the acute cases and the same is seen in the lungs. Parenchymatous degeneration of kidneys, myocardium, and liver is always present. Hemorrhagic areas in the urinary bladder are not rare. The bone-marrow shows conspicuous and constant changes. Especially in the femur and the humerus, the yellow marrow is partly or entirely changed into a dark brown red or black red mass. In the cases of a more chronic course, only several circumscribed hemorrhages in the fatty marrow are shown. The latter may appear to be normal, while in chronic and acute cases, the red marrow in the extremities is apt to show a red discoloration. The same is seen in the marrow of the vertebræ, ribs, etc.

The most frequent lesions met by Mack [50] are hem-

orrhagic areas in various organs, but most constant in connection with the intestinal canal. In some instances various lymphnodes were found to be edematous and hyperæmic, in others they were normal. The bone-marrow was found to be altered. Especially that of the humerus and femur showed a color darker than normal, with red areas in various parts. Histologically various changes were encountered, but nothing of a striking, characteristic nature. The presence of the petechiæ and ecchymoses in many of the viscera was a notable feature of one of the autopsies, but those changes were absent in the other. The lymphnodes were either normal or hyperæmic or edematous. Parenchymatous degeneration was present in the kidneys and liver.

Mohler [66] reports that animals dead with the disease are very emaciated and anemic. There is a marked absence of adipose. Subcutaneous and intermuscular edema and hemorrhages are frequently observed, although the absence of gross lesions is remarkable in many cases. Mohler regards the petechiæ of the heart as the most predominating and most constant lesion. The organ is usually enlarged. In some cases the lungs may be studded with petechiæ, while a serous exudate may be present in the thorax. The pericardial fluid is also generally increased. Peritonitis and a hemorrhagic condition of the intestine may be encountered. The liver is generally normal, although sometimes it presents a few areas of degeneration. The spleen, at times, is found to be enlarged and covered by petechiæ. The kidneys may appear normal or anemic and flaccid, while under the microscope they usually show a chronic parenchymatous degeneration. The lymphnodes may be enlarged and hemorrhagic.

Melvin [70] expresses the opinion that in a broad general way, it may be said that the blood is the real seat of the trouble, and that any pathologic changes observed in the circulatory system or in the viscera are dependent on this rather than that the lesions in the circulatory system are primary. This author further states that the atrophy and weakening of the muscles of the hind quarters, causing

Fig. 5. Experimental horse No. 637, 25 months after original infection.

Fig. 6. Experimental horse No. 658, 23 months after original infection.

the consequent staggering gait undoubtedly are the result of metabolic disturbances, which are inherent in the blood rather than in the muscles themselves.

Todd and Wolbach [75] found in one case that the marrow of the long bones was fatty and that it did not contain blood forming cells.

In order to permit a comparison between the autopsy findings of the observers mentioned and those made at the North Dakota Experiment Station, we hereby submit some notes referring to the latter. Those notes only refer to such field cases of which the infectiousness was proven by the fact that their blood conferred the disease to normal horses and to the findings in connection with those experimental cases.

No. 562. (Field case taken from an outbreak near Harwood, N. D.) At the time of death this animal was greatly emaciated and after the removal of the skin the lack of adipose and the pallor of the muscle tissues were much in evidence. There was a slight yellow tinge noticeable, in connection with the subcutaneous connective tissues. The peritoneum showed evidence of a chronic inflammatory process by the numerous fine threads of fibrinous exudate and slight adhesions. Those lesions are found on the parietal as well as on the visceral portion, but perhaps more marked in connection with the latter although at this time it is not thought that there exists any connection between those peritoneal lesions and "swamp-fever." They are probably the result of parasitic migrations.

The spleen is considerably enlarged (weight 5 lbs. 12 oz.). Its peritoneal covering is thickened over its entire surface, which is covered with an organized fibrinous exudate. The splenic lymphnodes are enlarged. The spleen substance is of a little more solid consistency than is found under normal conditions, but does not otherwise present any marked features. Microscopically the spleen structures do not present noticeable changes. The liver, aside from

being somewhat resistant to the knife, in its macroscopic aspect does not differ from the normal. Microtome sections of this organ reveal an apparently recent leucocyte infiltration of the interstitium. In several lobules, however, the round cells can also be seen between the cells of the parenchyma and around the vena centralis. Pigment deposits are common through the sections. The portal lymphnodes are hemorrhagically infiltrated and exceedingly friable. They contain considerable deposits of a brownish yellow pigment. The intestines show no marked changes. The cæcum contains a number of nematodes. A verminous aneurism is present on the anterior mesenteric artery. The aneurism is quite large and extensive and shows evidence of a chronic existence. It contains a few embryos of Sclerostomum. The kidneys are of normal size but pale upon section. The histologic examination of those organs shows in many instances a beginning of the thickening of Bowman's capsule, the glomerulus frequently occupying all the available space. The structure of the glomerulus is generally intact but here and there degenerative changes have taken place and between the debris leucocytes may be seen. The epithelium of the tubules is fairly intact, wherever it is not encroached upon by interstitial changes. Interstitial round cell infiltration is confined to certain limited areas. It is limited in extent and at no place is any evidence of induration noticed. The arterial walls are somewhat thickened. The lungs are normal in appearance with the exception of a pneumonia focus of about 15 c. m. diameter situated in the anterior portion of the right lung. The solidification seems to follow the larger bronchioles and presents numerous hemorrhagic infiltrations. The histologic picture is that of a broncho-pneumonia, which here and there shows evidence of indurative changes. In most of the sections the alveolar walls can scarcely be recognized, owing to the dense masses of leucocytes. They also fill the bronchioles, which in many places seem to have become atrophic or obliterated. In many of the vessels thrombi are present,

while near the periphery of the inflamed area hemorrhagic extravasation also fills the pulmonary alveoli. Unfortunate circumstances prevented the examination of the heart of this case.

No. 640. (Field case obtained from the neighborhood of Christine, N. D.) The cavader is very thin, the muscles are of normal color. The heart is normal in appearance but microscopically shows very slight evidence of granular degeneration and interstitial myocarditis which changes appear to be in their incipiency. The lungs are well contracted, presenting a few parasitic nodules under the pleura. There are also a few dark areas under the same membrane apparently due to a localized hyperemia giving rise to a somewhat mottled appearance of the regions involved. Several of the lymphnodes along the colon are enlarged and hyperemic. The intestines themselves are normal in appearance and contain a few specimens of Sclerostomum equinum. The liver is somewhat enlarged, dark in color and has its surface covered by fibrinous threads. Sections of this organ show parenchymatous degeneration, and interstitial hepatitis. Many lobules are infiltrated more or less with leucocytes and in some instances those cells are grouped in the form of small, round, narrowly circumscribed areas. There is a moderate degree of siderosis. The spleen is enlarged and shows numerous petechiæ on its surface. Microscopically, the trabeculæ seem thickened, while a considerable amount of pigment is in evidence. The kidneys are normal in appearance, and under the microscope, their parenchyma is fairly intact and interstitial round cell infiltration, although present, is insignificant in extent. The vessel walls are slightly thickened.

No. 635. (An experimental case infected by an injection of blood from No. 640.) The cavader is emaciated and there is a great reduction of adipose. The skeletal muscles have a normal color. There is a general peritonitis with a fibrinous exudate. The colon and cæcum are hyperemic. The mucosa of the colon is intensely inflamed or hemorrhagically infiltrated and a large

quantity of hemorrhagic exudate is found in the lumen of the gut. Sections of the wall show it to be densely packed with leucocytes and blood debris, while numerous small necrotic areas are noted in the sub-mucosa. The floating colon is stippled with petechiæ. The omentum is densely inflamed. A small parasitic aneurism is noted in the anterior mesenteric artery and contains a few worm embryos. The liver is enlarged, hyperemic, very friable and histologically presents evidence of a considerable amount of fresh interstitial leucocyte infiltration. Many round cells are seen within the lobules. There is a moderate degree of siderosis. The spleen is about six times its normal size, its pulp very dark and soft. Sections of the organ show blood engorgement and a great amount of siderosis. The trabeculæ are not changed. The lungs are poorly contracted with a slight fibrinous exudate on the pleura and the heart is soft, flabby, par-boiled in appearance, but has its microscopic structures fairly well preserved. The kidneys are soft, friable and richly interspersed with miliary abscesses. A purulent pyelitis is in evidence. Microscopically, there is a considerable amount of interstitial nephritis, with here and there areas of dense acute inflammation (miliary abscesses). Bowman's capsule is considerably thickened. The epithelium is generally intact when not encroached upon by extratubular changes. The tubules themselves contain a small amount of granular debris, but there is no evidence of tube casts.

No. 744. (An experimental case infected by means of the blood of experimental case No. 636.) The well developed adipose presents a yellow tinge. The abdominal cavity contains about one-half liter of fluid. The peritoneum is bluish white in color, smooth, shiny and transparent. In the liver the lobules present a more than usual distinct demarcation, its capsule is tense and its borders rounded and of a light blue color. The spleen is apparently normal. The capsule of the kidney is tense and the tissue friable. Stomach and intestines as well as their contents present nothing abnormal. The left lung is pink in

color and slightly emphysematous on ventral border. The right lung is dark blue in color and somewhat edematous. The heart tissue is quite firm but of a pale color.

No. 855. (An experimental case infected by means of the blood of experimental case No. 636.) The carcass is very thin and there is a lack of subcutaneous, sub-mucous, sub-serous, and omental fat. The peritoneum is normal and contains a small quantity of fluid. The kidneys and pancreas are apparently normal, but the former show upon microscopic examination, a few, very slight and probably recent interstitial round cell infiltrations. The glomeruli are somewhat congested and a few tubules contain hyaline casts. The bladder contains about a quart of normal urine and has a normal mucosa. The external surface of the spleen presents evidence of a slight adhesive peritonitis, and shows numerous ecchymoses confined to its capsule. The liver aside of a pronounced adhesive peritonitis on its surface, has a normal appearance. The stomach contains about ten "bots" but is otherwise normal. The small intestines are normal. The mucosa of the cæcum showed a number of Sclerostoma, also three nodules (parasites) between the serous and muscular coats. The first and second sections of the large colon showed myriads of small round worms (probably S. tetracanthum) mixed with the contents. The third section showed several ulcers in the mucous coat, which were of a probable parasitic origin. The different lobes of the lungs contained numerous small nodules principally underneath the pleura, but few were scattered through the lung tissue. The pleura is normal. The heart has a normal appearance and does not show any microscopic lesions. All lymphnodes examined are normal. Bone-marrow changes are pronounced especially in the femurs. The proximal cancellated bone tissue is densely hyperemic and a good part of the fat marrow shows a change to lymphoid structure.

No. 857. (An experimental case infected by means of the blood of experimental case No. 636.) The car-

cass is poor, has very little adipose, but the muscles are normal in color. The small intestines and floating colon are normal, but the mucosa of cæcum and large colon is inflamed over a considerable area. Sclerostoma are present but not in large numbers. The spleen is normal but shows slight siderosis. The naked eye appearance of the kidneys is normal with the exception of small calcareous deposits being present in the medulla just outside the pelvis. Microscopically the kidneys show some capillary engorgement and areas of interstitial nephritis, but the parenchyma is fairly intact. In the region of the diaphragm and liver there is evidence of a considerable chronic peritonitis in the shape of numerous adhesions. Otherwise the gross appearance of the liver is normal. Microscopically the organ shows interstitial and intralobular round cell infiltration and some parenchymatous degeneration. The bone-marrow especially that of the femur shows areas of a dark red discoloration, the remainder being pale and very fatty.

No. 867. (An experimental case infected by means of the blood from experimental case No. 636.) The subcuta contains a yellowish, gelatinous substance, especially well marked on ventral portion of abdominal wall. There is marked evidence of an old adhesive peritonitis, both parietal and visceral. There is no excess of peritoneal fluid. The spleen is slightly enlarged and shows numerous hemorrhagic pin-head areas beneath capsule. Microscopically there is marked siderosis and slight hemorrhagic engorgement. Splenic lymphnodes are slightly hemorrhagic. The small intestines show a distinct, acute enteritis throughout, with the exception of the first ten feet of the jejunum. The bladder is devoid of urine, but contains a couple ounces of yellowish, brown, sandy sediment. There is also evidence of a slight cystitis. The kidneys contain a mucoid deposit, closely adherent to the pelvis, otherwise they appear to be normal to the naked eye. Sections, however, reveal pronounced interstitial lesions but the parenchyma is well preserved. The large and floating colons

are normal but the mucosa of the cæcum presents slight petechiæ near the apex. The stomach contains a small amount of ingesta and presents a well marked gastritis. The visceral lymphnodes show a slight hemorrhagic condition. The liver has a normal size. Its capsule shows well developed fibrinous appendages, some penetrating into the liver structure. The border of right lobe shows a considerable hemorrhage. Histologically, this organ shows an interstitial hepatitis, well marked and of recent origin. There is a slight siderosis and the parenchyma is fairly intact. Both layers of the pleura show adhesive pleuritis. The lungs are collapsed, the right one showing hypostasis. The bronchial and mediastinal lymphnodes are hemorrhagic. The pericardial sack contains about 500 c. c. of a yellowish, red fluid. It is slightly adherent to the diaphragm. The myocardium shows small petechiæ, while there are very marked ones on the endocardium. The bicuspid valve has a marked hemorrhagic area extending throughout the entire valve. Microscopically the myocardium shows a beginning of brown atrophy with here and there a slight evidence of interstitial myocarditis. The marrow of the proximal extremity of the left femur is dark red in color, the marrow cavities being filled with an abundance of dark red pulp. The marrow of the distal extremity is pale, the pulp apparently being replaced by a fatty debris. The place normally occupied by the yellow marrow shows a large area of hemorrhagically infiltrated tissue. This dark red part shows an arrangement in foci. Each such focus is surrounded by a bright red zone, which is somewhat separated by an intervening light colored strip from the dark red structures. Of the right femur the upper extremity is like that of the left, while the lower also resembles the corresponding part of the left bone.

No. 902. (An experimental case infected by means of blood from experimental case No. 855.) The cavader is thin. The peritoneum shows a slight yellow tinge and is quite pale. The spleen is normal in size,

but shows numerous slight, sub-capsular hemorrhages. The borders are slightly rounded and the organ shows a rather tense appearance. Splenic lymphnodes are somewhat edematous and enlarged. The liver is normal in size and appearance with slight adhesions to the diaphragm. The small intestines appear to be normal with the exception of several ecchymotic areas on the mucosa. The large colon presents a normal appearance. Its contents show several specimens of Sclerostomum equinum. There are numerous ecchymotic spots in the region where the parasites are found. Several small nodular enlargements were to be seen in the mucosa. The cæcum contained a markedly hemorrhagic area. The kidneys are apparently normal. The bladder contained about 500 c. c. of a straw-colored viscid urine. Its mucosa is slightly congested in the ventral portion. The heart muscle is quite firm and has a good color. The left ventricle presents a slight ecchymosis of the endocardium. A small hemorrhagic area is found on the auriculo-ventricular valve. The lungs show no changes and collapsed completely when the thorax was opened. The bone-marrow shows but very few hyperplastic areas.

No. 903. (An experimental case infected by means of the blood of experimental case No. 873.) The general condition of the carcass is good and all adiposa are well developed. The peritoneum shows pronounced visceral adhesions, especially on liver and spleen; the peritoneal fluid is normal. The right kidney is apparently normal; the left one is considerably congested (antemortem). The bladder is normal and contains 200 c. c. urine of an orange-yellow tinge (S. G. 1046, neutral reaction, contains a trace of albumen). The spleen is normal, with the exception of the adhesions noted above. The splenic lymphnodes are enlarged and hemorrhagic. The liver presents no changes, aside of the adhesions previously mentioned. The mucosa of the small intestines presents numerous fresh hemorrhagic areas and that of the cæcum, a few small ulcers, evidently of a parasitic origin. The first division of the

large colon contained a great number of helminths (Sclerostoma). The apparently normal stomach contained a few "bots." Lungs and pleura are normal. No changes are seen in connection with the heart, but a few projecting calcareous concretions are attached to the intima of the common aorta just behind the valves.

Nearly all the visceral and body lymphnodes were slightly enlarged and hemorrhagic. Hyperplasia of the marrow is seen in the femurs and humeri while the proximal cancellated structures either are hemorrhagic or marked by small dark areas.

No. 642. (A field case purchased in the vicinity of Fargo, N. D.) There is an almost complete absence of subcutaneous fat. The musculature, although reduced in volume was normal in color. The lymphnodes lying on the cæcum are enlarged and surrounded by hemorrhagic areas. The mesocolic lymphnodes are enlarged and soft, but the mesenteric nodes are not involved. In the cæcum there are a few parasites (Sclerostoma) and at certain places the mucosa is ecchymotic, while near the apex, there are quite a few parasitic nodules in the mucosa. The kidneys are slightly enlarged, soft, pale and friable, but sections taken from those organs show a slight degree of interstitial involvement, a shrinking of the glomeruli and an intact epithelium. The spleen is normal in size, thin and flabby. Cutting through the organ, it seemed as if the spleen pulp was reduced, while the connective tissue elements were more conspicuous. The liver is normal in appearance. The lungs are poorly contracted, while the right anterior lobe is somewhat emphysematous. The heart was pale and soft, and its cavities filled with ante-mortem clots. The peritoneum showed evidence of chronic inflammation with an organized fibrinous exudate, more especially near the diaphragm. The anterior and posterior mesenteric arteries showed small aneurisms. In the bladder was found a small quantity of urine (S. G. 1025, acid in reaction and free from albumen).

No. 639. (An experimental case infected by means

of blood from experimental case No. 637.) There is a reduction in adipose. The muscles are generally of normal color. The spleen is about twice the normal size, shows some infarcts and microscopically, reveals a moderate degree of siderosis. The kidneys are paler than normal and the naked eye appearance leads one to think of parenchymatous degeneration. Microtome sections, indeed, reveal the presence of a naked congestion of the medullary vessels. There is a very marked interstitial nephritis, accompanied by shrinkage and degenerative changes of the glomeruli, with thickening of the capsule. In a few of the convoluted tubules there are some signs of parenchymatous impairment and many of the straight tubules contain casts. In the liver interstitial inflammation is marked and a great number of leucocytes have infiltrated into the lobules. Siderosis is apparent throughout and the parenchyma shows some slight fatty changes. The large colon shows a few sub-peritoneal nodules, while a few specimens of Sclerostonum equinum are contained within. The anterior mesenteric artery shows a large verminous aneurism. The lungs are well contracted and normal, with the exception of a small area of solidification which has a softened area in the center. The heart looks normal, but microscopically a very slight degree of interstitial myocarditis can be detected. The mucosa of the nasal septum presents a large, gangrenous area. This ulcertation was apparently due to a deep necrosis and extending clear through the septum involves the mucosa of both sides to the same extent and in the manner that the affected mucous surfaces were directly opposite one another. A large eschar is still intact in the ulcer and represents the gangrenous mucosæ and cartilage. The edges of the ulcer were rather smooth and at places occupied by a softened mucosa, apparently becoming gangrenous by continuity. There is nothing to indicate malleus and a conscientiously carried out bacteriologic research confirms this statement. The sub-maxillary lymph-

nodes were somewhat enlarged, but neither softened or purulent.

No. 743. (An experimental case infected by means of the blood from experimental case No. 638.) The lungs are inflated and show numerous slight adhesions between the pleural layers. There is no liquid exudate in the cavity. The anterior half of the lungs are filled with solidified foci, the cephalic lobe being entirely solid. The ventral border also solid to a width of about two inches. The ventral border of the posterior lobe also contains many solidified foci and presents numerous ecchymoses beneath the pleura. Along the ventral border of the posterior lobe there are numerous small necrotic areas. The pericardium contains about one liter of a straw-colored exudate. The right side of the heart shows marked ecchymoses and the auricle contains a large ante-mortem clot, which extends into the ventricle. The left side shows no changes. Sections of the heart reveal a general cloudy change of the myocardium. The liver has a weight of twenty pounds, is somewhat congested and firmly adherent to the diaphragm. The posterior surface was likewise somewhat adherent to the stomach, which organ showed a corresponding inflammation on the external surface, but its mucosa showed no lesions. Liver sections showed a well advanced parenchymatous degeneration and some siderosis. The intestines show nothing abnormal. The kidneys and bladder appear to be normal, although sections of the former show slight interstitial changes throughout. There is a small degree of arterial thickening, while the glomeruli are enlarged filling out the entire capsule. A very slight degree of parenchymatous damage may be mentioned. The spleen is quite flaccid, shows some necrotic areas and a moderate degree of siderosis.

No. 723. (An experimental case infected by means of the blood of experimental case No. 638.) The cavader is poor, but not emaciated, the muscles have a normal color. The spleen has a weight of four pounds, six ounces, and shows small hemorrhages under the

capsule. Heart, kidney, liver, intestines and lymphnodes are normal in appearance. The marrow of radius and tibia is pale, deficient in coloring and is quite fatty. The same conditions prevail in the lower extremity of the humerus and femur, but the upper extremity of the femur is lightly red. The cancellated tissue of the proximal extremity of the humerus has a mottled appearance.

No. 816. (An experimental case infected by means of the simultaneous injection of the blood from experimental cases Nos. 637 and 638.) The subcutaneous connective tissues are poor in adipose, lemon-colored, while the musculature has a salmon color. All the mucosæ are blanched. The lungs are inflated and show petechiæ on pleura. The bronchial and mediastinal lymphnodes are normal with the exception of a certain degree of anthracosis. The heart has a par-boiled appearance with pronounced ecchymosis of the endocardium of the left ventricle. The kidneys are of normal size, but show dark spots on surface and their substance is rather friable. The large intestines show very numerous petechiæ, which are also visible through the mucosa. The liver is normal in size and shows a fibrinous exudate on the surface. The spleen is slightly enlarged and has a few petechiæ on the surface. Petechiæ are also seen on small intestines, mesentery and bladder. In the upper end of the femurs, the bone-marrow is red for a considerable proportion and this red marrow is sharply demarcated from the yellow parts. The marrow of the lower part is of a yellowish-white color and edematous. The humeri and tibiæ have a marrow like that seen in the lower part of the femur, but in that of the lower part of the radii, there is still a pink color present, although it is very pale.

No. 920. (A field case purchased in the vicinity of Kelso, N. D.) The carcass was poor but not emaciated. The quantity of adipose is small. The peritoneum is apparently normal and contains about one liter of peritoneal fluid. Pancreas and kidneys are normal in appearance. The bladder looks normal and contains about 200 c. c. of amber-colored urine

(S. G. 1015, reaction strongly acid, albumen present). The spleen is of natural appearance but the splenic lymphnodes were enlarged, showing a hemorrhagic lymph-adenitis. The liver is enlarged to one and one-half the normal size, showing a marked adhesive peritonitis and pronounced siderosis. Under the microscope, the interstitial changes prove to be pronounced, there are granular changes in the parenchyma, while pigment deposits are seen throughout the sections. The portal lymphnodes are enlarged and hemorrhagic. There is a well defined verminous aneurism on the anterior mesenteric artery. The small intestines contained a small quantity of semi-fluid ingesta, through which were scattered about a dozen specimens of Ascaris megalocephala. The cæcum and large colon contained a large quantity of semi-fluid ingesta. The cæcal mucosa showed a few specimens of Sclerostoma. The floating colon is normal to the naked eye. The mesenteric lymphnodes were greatly enlarged and hemorrhagic. The normal stomach contains about six large "bots" and about a dozen smaller ones. The gastric lymphnodes present the same features as the mesenteric ones. The parietal pleura shows a well marked ecchymosis, the result of a pleuro-pneumonia. Both lungs show a well marked lobar pneumonia, the left being very extensive, taking in the entire ventral half. The pulmonary artery showed a marked thrombus. The bronchial and mediastinal lymphnodes are enlarged and hemorrhagic. The pericardial sac contained about one quart of fluid. There is subepicardial ecchymosis. The myocardium is pale, flabby, and apparently degenerated. Subendocardial ecchymosis is in evidence. The right heart is filled with an ante-mortem clot. The marrow changes are the same as in the other cases; there is hyperplasia of the fat marrow and hemorrhagic areas are noted in the other parts of the bone.

No. 930. (An experimental case infected by means of the blood from experimental case No. 919.) The cavader is thin and presents a scant amount of subcutaneous, sub-serous, sub-mucous, and omental fat. The

peritoneum is apparently normal with the slight exception of some adhesions of the viscera. Liver, spleen and kidneys are seemingly normal. The bladder contained about one liter of lemon-colored urine and has a normal mucosa. (Urine: S. G. 1030, reaction neutral, albumen present). The stomach and small and large intestines are normal. There is a colony of "bots" in the stomach and the large intestines contain contain a few Sclerostoma. Lungs and pleura are both normal. The heart presents slight subendocardial petechiæ and ecchymoses, but is otherwise normal. The lymphnodes are apparently normal with the exception of the mesenteric ones, which are slightly edematous. The marrow of the bones shows some hemorrhagic infiltration and a few well defined hemorrhagic areas.

No. 921. (Field case purchased in the vicinity of Shelly, Minn.) The carcass is thin and shows a marked edema of the sheath, the lower abdominal and thoracic walls, between the fore legs and on the inner side of the thighs. The adiposa are reduced in volume and the subcuta shows a slight lemon color. The fairly well developed musculature suggests a lemon color. The peritoneum presents numerous fibrinous adhesions to the viscera and contains about one liter of a dark thin hemorrhagic fluid. The stomach is normal; there are not "bots" and the gastric lymphnodes are normal also. The small intestines offer no changes, but their lymphnodes are hemorrhagic. The large intestines are normal throughout. The heart is enlarged, flabby, and has a par-boiled appearance. There was about one liter of dark hemorrhagic fluid in the pericardial sac. There are subendocardial petechiæ near the columnæ carnæ of the left side and the auricles are filled with large chicken-fat clots. The kidneys are normal in size, but very pale, probably the seat of a considerable parenchymatous change. The renal lymphnodes are enlarged. The liver shows a post-mortem change, but is normal in size. The spleen is slightly enlarged and shows petechiæ under its capsule. The bladder contains about one liter of urine (S. G. 1012, and acid reaction and contains a

trace of albumen). The upper part of the bone-marrow of the femur is of a deep hemorrhagic red. The marrow cavity contains fat marrow to one-half of its capacity, while the other half contains a hyperplastic red marrow. The extremities of the humeri are extremely pale and here and there show small hemorrhagic areas. The fat marrow also shows a few hyperplastic areas. In the tibiæ and radii, a uniform lemon color is observed.

No. 924. (An experimental case infected by means of the blood of field case No. 921.) The carcass is in fairly good condition. The panniculus adiposis is well developed and submucous, sub-serous, and omental fat is not materially reduced. The peritoneum, with the exception of some slight adhesions between liver and diaphragm, is normal throughout. The kidneys are both congested and the bladder contained about two liters of urine (S. G. 1020, neutral reaction, containing a trace of albumen). The liver has a normal appearance as well as the spleen. In the small intestines there are diffusely spreading hemorrhages, the duodenum showing a decided congestion. In the cæcum and large colon, the mucosa is almost a continuous hemorrhagic mass and the vessels of the sub-mucosa are heavily injected. With the exception of a few subendocardial petechiæ, the heart is normal for so far as naked appearances go. The lungs and pleura are normal. There is marked hyperplasia of the bone-marrow of the femurs, but in the other bones, this condition is less marked.

No. 934. (An experimental case infected by means of the urine of experimental case No. 924.) The cadaver is in fair condition, although there is but a slight amount of subcutaneous, sub-mucous, sub-serous and omental fat. The kidneys are slightly enlarged and show slight capsular and parenchymatous hemorrhages. The bladder contains about 500 c. c. of urine (S. G. 1024, reaction sharply acid, contains a considerable amount of albumen). The spleen is considerably enlarged and the pulp showed a blackberry jam-like color and consistency. The liver is

slightly enlarged and wholly free from adhesions. The mucosa of the stomach showed marked ecchymoses. With the exception of a couple of parasitic nodules, the small intestines are apparently normal. The large intestines present a few parasitic ulcers to which a pair of specimens of Sclerostoma were attached. The lungs were normal. The heart shows subpericardial ecchymoses, myocardial hemorrhages and the endocardium in all compartments of the heart is covered by hemorrhagic areas. Most of the visceral lymphnodes and especially those of the spleen are enlarged and slightly hemorrhagic. The bone-marrow changes in this case are slight and consist principally of the presence of small hemorrhagic acreas.

Reviewing our findings made at autopsies and those reported by other investigators, it would seem that none of the lesions are such as deserve to be regarded as pathognomic. There are no changes which unmistakably bear the brand of identity. Almost any of the changes met with may be found in association with other diseases and in themselves are of but little diagnostic value, and hence they can only be used diagnostically as a certain link in a chain of evidence furnished by clinical, epidemiologic and experimental data. The features deserving the most attention are no doubt the petechiæ and ecchymoses of certain structures, lymphnode involvement, interstitial and parenchymatous lesions of certain important viscera, albuminuria, and the alteration of bone-marrow and the blood. We have refrained from discussing the latter in connection with the pathologic anatomy of the disease, preferring to do so when dealing with the clinical features of the disease. It is possible that the bone-marrow lesions constitute a valuable aid in the post-mortem diagnosis of the disease, but in view of the fact, that we know, comparatively so little of bone-marrow changes in connection with other diseases of the horse, or even under varying normal conditions, we would rather suspend judgment relating to their diagnostic value

until the matter has been looked into more fully. The writers contemplate making some observations in regard to marrow changes in various conditions and hope to be prepared to place a proper value on those seen in "swamp-fever" in the course of the coming year.

Studying the clinical manifestations of the disease, we meet with a more or less constant series of symptoms, which are somewhat characteristic. Taylor [7] speaks of "swamp-fever" as an intermittent fever somewhat analogous to surra and characterized by anemia. The one case described by him has a slight attack of fever, was rested up and appeared at one time to have recovered. When driven, however, it appeared so sluggish, that it staggered when led into the stable. In addition to the staggering gait, the animal showed signs of laminitis. The weakness of the hind quarters remained a prominent feature in this case, but the appetite showed but little impairment. The animal soon commenced to lose flesh, the abdomen became tucked up. Taylor also speaks of a discrepancy existing between the height of temperature elevation and the increase in the number of pulse beats and regards it as "one very diagnostic symptom." In Taylor's case the mucosa became paler than normal, having a yellowish tinge, which became more evident as the disease advanced. The animal improved and relapsed several times.

The patients observed by Köpke [8] stood with their heads down in the manger and showed a rather capricious appetite. The conjunctiva was conspicuously pale without showing any yellow tinge. In the beginning of the disease, the pulse was relatively strong, but more frequent than normal (50-60 p. m.). The most pronounced symptom was the high body temperature which in nearly all cases was elevated from the beginning. (105.5°-106.7°F.). In the further course of the disease, the fever remained almost constant for eight to fourteen days. The heart function decreased in force, the pulse becoming weaker and its fre-

quency ranging from eighty to one hundred beats per minute. At the same time the pallor of the conjunctiva became more marked. In the cases running a favorable course, the temperature receded gradually to normal in from six to eight days, and the animals made a complete recovery in from two to three weeks. In the more severe cases also, the temperature came down in from ten to fourteen days, but only to 102.2° F. to 101.1° F. The patients then became more lively, but the pallor of the mucosa and the cardiac weakness persisted and after an apparent improvement of from ten to fourteen days, another febrile attack with the usual accompaniments had to be sustained. This would happen three or four times within two or three months. During this time, the animals would lose considerable flesh. In many cases a severe diarrhea would set in. During the last stages of the disease, the animals would sway considerably with their hind parts and could no longer maintain themselves in the standing posture. As an accompaniment of the complete anemia of the mucosa and a high degree of cardiac weakness, marked edema of the dependant parts of the body would make its appearance and the animals, after an illness lasting from four to eight weeks, would succumb in a very emaciated condition.

As described by Torrance [9] the disease is essentially a fever of the remittent type characterized by progressive anemia, gradual emaciation in spite of a good appetite, edema, weakness and loss of power in the hind legs. Weakness is the earliest symptom, which is closely followed by uncertainty in the movements of the hind legs, which causes the animals body to sway the hind quarters. The appetite continues normally or is even increased. The pulse gradually assumes a greater frequency, running from 50 to 70 beats per minute and presenting a peculiar thrill as if the vessel were only partly filled. The temperature ascends to 103° F. or higher and shows considerable irregularity. The animal now becomes too ill for work and may receive some form of treatment. In spite of apparent improvements, which how-

ever, are only temporary, the animal becomes thinner, the mucosa becomes pallid on account of the profound anemia, while the edema of the dependant parts of the body makes its appearance. The pulse becomes more rapid and weak and the heart action labored. The skin is dirty and greasy to the touch and the animal has polyuria. Torrance also met sometimes with more acute cases in which the symptoms are presented in a more aggravated form and the animal dies in two or three weeks. The red blood count of Torrance's cases went as low as two millions. The corpuscles retain their form well and this author never witnessed marked poikilocytosis, although there has generally been seen a small proportion of megalocytes and microcytes. There was a marked increase in the clotting properties. In one or two acute cases, Torrance observed, what appeared to be debris of red corpuscles floating in the plasma. The granules were formless, irregular in size and had no power of motion.

Brimhall, Wesbrook and Bracken [10] write that in the Minnesota disease the onset was insiduous, but once developed, it was accompanied by a general weakness, slowly progressive, loss of flesh, periods of fever, followed by times of apyrexia, gradual emaciation, staring coat, a ravenous appetite, and sometimes by polyuria, as the most prominent symptoms.

The following symptoms are described by Rutherford [11] as the most prominent: progressive emaciation, edematous swellings of the dependant parts of the body, progressive pallor of the visible mucosa on which petechiæ may be occasionally seen. The peculiar soft, flat pulse is regarded as almost diagnostic, while the periodic rise and fall of the body temperature, ranging from normal to 105° F. is regarded as the most important of all symptoms. The appetite, while capricious, generally remains good until near the last. Lack of muscular control is common in the more advanced cases, but Rutherford ascribes this more particularly to the

general weakness. Polyuria is frequently seen in the latter stages.

The ten cases reported by Béghin [22] all commenced by losing their appetite, then they became dull and still later they acted as if paralyzed. All animals sustained a considerable loss of flesh. They showed swelling under the chest and belly, while in the geldings the sheath became swollen also. The fatal cases went on to the extreme emaciation. The one case actually seen and examined by this author himself was very poor in flesh, hide-bound, with a staring coat and stood with its head down. The appetite was diminished and irregular. The mucosa are pale. The pulse is small and very much accelerated. The artery is soft, but the beats are regular in spite of those changes. There is a slight edema under the chest and the heart beats violently. A marked systolic murmur can be detected. The respiration is more frequent than normal, but there is neither cough nor discharge. The temperature is 103° F. and weakness is a marked feature, the animal having difficulty in moving the hind legs.

The fundamental observations made by Carré and Vallée [23] deserve to be quoted quite fully. Their investigations led them to recognize three clinical forms of the disease; namely, the acute, subacute, and chronic types.

The acute type sets in suddenly. The animals become soft and lazy when at work. The least effort at pulling or speed causes the animals to blow or in some cases even to fall down. The appetite fails and the food is taken slowly. The conjunctiva is injected, edematous, yellowish with a red ground-color. Petechiæ of a translucent aspect are frequently observed. They are pink red, brownish or purplish in color and from 10-20 m. m. in diameter. A flow of tears is quite often seen. The temperature rises to 104°, 105°, sometimes even to 107.5° and generally keeps up until the end. The animals hold the head in a fixed position, the nostrils are dilated, and the facies reminds one of tetanus. Evidence of enteritis with diarrhea makes its

appearance. The fæces are often reddish in color and would lead one to believe that they contain blood, if the spectroscopic examination were not negative in its results. In certain cases, however, the fæces are streaked with blood.

No respiratory disturbances are encountered. The pulse is soft, quite frequent (60-90 beats per minute). The cardiac sounds are accentuated. Marked edema is not encountered, only a simple swelling of dependant parts. The urine is abundant, generally deep in color and always containing albumen. In certain cases the albumen amounted to 14 grammes per liter. As a general thing urine does not contain blood corpuscles or hemogoblin, but granular and epithelial casts may be present.

The animals sway or take the position of a foundered horse. Movements are painful and the wabbling gait is a marked feature. Getting up is difficult, there being present a clear paresis of the hind quarters. There is a progressive emaciation and the extreme weakness and paresis of the hind quarters may be expressed by incontinence of urine. The coat is staring and the hairs of the mane and tail can be pulled out with extreme ease. Pregnant mares commonly abort. In this form of the disease, the course ranges from five to fifteen days, more often about eight days.

In this acute type, the changes of the blood are but little pronounced. There are no signs of anemia. Nevertheless, the blood coagulates but slowly. There is a rapid separation of the corpuscles, which have a brownish red tinge and which agglutinate, massing themselves in small agglomerations floating in a highly colored plasma, which frequently is opalescent.

The subacute type presents the same features as the acute one, but they are more or less attenuated and differently associated. Clear cut remissions are a distinguishing symptom of this form, a remission, which, when anemia does not happen to become apparent, would lead one to accept a real recovery from the disease. Carré and Vallée even saw cases which went along for eight months without

presenting fever. Such recoveries, however, are more apparent than real, as the urine still shows albumen upon analysis and the conjunctiva is edematous. Blood examination reveals a marked anemia. The slightest work causes the animals to blow, while the heart beats become fast. The animals sweat abundantly and sometimes drop in an exhausted condition. In this form the duration of the disease varies very greatly, ranging from a few weeks to several months.

The chronic type is characterized by the symptoms of an extreme anemia. The patients become soft, lazy, the appetite indifferent and capricious, the coat rough and staring, while the hair of tail and mane can be pulled out with ease. The mucosa are pale and the temperature is most frequently normal; there are intermittent attacks of fever, but they are not frequent. The animals are soft and weak. The pulse is soft, frequent, unequal and the artery is flabby. Sometimes a transitory diarrhea is observed. Edema of the dependant parts is seen and the gait is wabbly, stumbling, and arising is difficult. The urine is abundant and more often contains albumen. The anemia is progressive, the blood coagulating poorly, forming a soft small clot. Sometimes, even it is difficult to check hemorrhage in patients. After more or less frequent remissions the animals die, either in one of the exacerbations or succumb to the final stage of anemia.

Changes in the blood are never absent and pertain to both plasma and formed elements. The plasma is but very little coagulable, it is over-colored, of a deep yellow or greenish tint, very often dichroic, especially so in the acute cases and during the acute exacerbations of the chronic cases. The erythrocytes are friable and readily agglutinated. They are irregular in size. They are poor in hemoglobin and fragile. Crenation is very commonly found. The red corpuscles, after being stained with the basic aniline dyes enclose a body comparable in shape and color to a piroplasma.

The red cell count varies according to the type of the

disease and the time of the examination. In the acute form, when the signs of anemia are masked by other symptoms, there already is a notable reduction in the number of cells. From the tenth or fifteenth day, the cells fall from one and a half million to two millions below the normal figure (about seven millions). At the time of death in the acute cases, the count only runs to four millions, in the subacute cases about two to two and a half million, and in the chronic cases the count will range from two to four millions, according to the condition of the patient. In some cases it will fall to one million. The leucocyte count does not show great deviations, there commonly being a slight leucopenia.

As seen in Nebraska by Peters [26] the disease is initiated by a recurrent fever, which is followed by weakness. The mucosa of mouth and eyes become very pale. A staggering gait is noticed as the disease progresses. The appetite is retained but the emaciation is progressive. The temperature becomes higher, ranging between 103° F. and 106° F. Temporary improvements were observed. Diseased horses usually live from two to three weeks, although Peters mentions some cases, which lived three months. Edema of the dependant parts of the body and petechiæ on the mucosa were observed. Polyuria is spoken of as a symptom of a serious condition. The blood count in field cases amounted to 1,800,000 to 2,000,000 red cells per cubic m. m.

Ries [27] does not regard the clinical picture as absolutely uniform. In certain subjects the disease runs its course without fever. The appetite is retained or diminished, while the strength becomes reduced. There is shortness of breath and the animals break out in sweat. Along with the presence of those conditions the anemia already is quite evident; the mucosæ are pale, white, and the pulse is weak, thready, while the heart sounds have acquired a metallic ring.

In other cases no alarm is taken before the appearance of the edema of the abdomen and the limbs. This form is especially seen during summer and early fall and on farms where there is a short supply of oats. In another form of

the disease, albuminuria is seen, aside from a notable increase in the rate of respiration and a fever which fluctuates around 104° F. This albuminuria may be pronounced and exhausting in some cases, while in others the albumen only occurs in traces. The animals continue to eat but waste, as it were, before one's eyes. The mucosa are not yet blanched but become paler. The eyes present a peculiar aspect, they are bright, feverish with the bulbs retracted, the eyelids participating in this movement. In the advanced stages, the patients move about with difficulty, sway to and fro, and threaten to fall. The limbs become edematous as well as the lower parts of the body. The horse no longer lies down, but when he does so in from five to ten days, he goes down for good.

The symptoms named by Charon [34] are a rather sudden loss of flesh, softness when being moved, swaying of the hind quarters, a pale yellow color of the conjunctiva, muffled heart sounds, arterial depression, venous pulse, a well marked reduction of the blood cells and an irregular but rather high temperature. The appetite, at first capricious, becomes better, the animal eating abundantly. The hair coat was not bad and the conjunctiva yellowish, but neither infiltrated nor oily. In Charon's case there was no edema of the sheath, the abdomen, the chest or the hind limbs. At no time was there polyuria or albuminuria.

Ostertag [35] elicited the following description of some cases from an owner, who in the space of five years has lost some twenty horses with the disease. The first phenomena noticed was a knuckling of the hind legs, while in the course of eight days, the animals would be tired and unfit for work. They would lose flesh rapidly and in a high degree. The mucosæ would be yellowish-white and periodically the patients would show a high fever. A few animals also presented edematous swellings of the lower part of the chest and the sheath.

Among the most conspicuous symptoms, Francis and Marsteller [42] mention: progressive emaciation without any

apparent cause, polyuria and a peculiar weakness of the hind quarters, which has given the disease the local name of "loin distemper." There are periodic attacks of fever lasting several days to a week or more; the temperature ranging from 103° F. to 107° F., and being followed by a drop to sub-normal, ranging from 95° F. to 98° F. There are remissions in the course of the disease, even apparent recoveries. The mucosæ are very pale, often of a yellowish or rusty color, while petechiæ may be observed on the conjunctiva as well as a dribbling of a bloody serum from the nostrils and anus. The skin becomes dirty, the eyes are sunken and there is a dribbling of urine with relaxation of the sphincters. Notwithstanding all this, the animals will eat ravenously until the last. The authors did not observe any nervous disturbances. They found anemia to be present. Some of the erythrocytes being abnormally large, while others were small. Nucleated cells were but rarely encountered.

Hutyra and Marek [69] recognize two forms of the disease. The acute form is ushered in by well marked signs of sickness. There is a pronounced dullness and general weakness, so that the animals easily tire and even fall during exertion. The weakness is most plainly seen in the hind quarters. Fever is simultaneously observed. This reaches its maximum height in from two or three days (104°-107°F.). It may remain constant until death or resume a resistant character. The pulse frequency is increased to 60-90 beats per minute, while it becomes weaker and softer, the heart impact being increased in strength. The conjunctiva is somewhat puffy, diffusely red in color, with a yellow tinge and on which irregular hemorrhagic areas are not rarely seen. The nasal mucosa also is reddened and shows petechiæ, especially on the lateral wall of the nasal cavity. Not infrequently, this is accompanied by a scant serous, sometimes reddish nasal discharge. A cough is sometimes noticed. There frequently is diarrhea, the dung sometimes being tinged with blood. Urine is frequently voided and

contains albumen, of which the quantity may amount to 1.5%, as well as granular and epithelial casts. The appetite is suppressed from the beginning. A progressive loss of flesh, which may make such a headway, that in a few days the animals lose from one-fourth to one-third of their body weight. Slight edematous swellings of the dependant parts of the body were only rarely observed. The coagulability of the blood is lessened, the serum appears dark yellow or even somewhat greenish and dichroic. The number of erythrocytes lessens the duration of the disease. The duration of the acute cases ranges from 5-15 days, but may be extended to from three to four weeks.

Hutyra and Marek's chronic form of the disease is characterized by periodic attacks of fever and evidence of anemia. The occurrence of albuminuria is not constant and when present during the febrile attacks it is only slight. In this form edematous swellings are not rare. Nutrition is reduced from the beginning or remains satisfactory in those cases which are marked by a slow progress of the disease. Loss of flesh occurs sooner or later. The duration of this form of the disease extends from one to several months and may drag even over a few years.

Mack [50] speaks of acute, subacute and chronic types of the disease and states that they are not separated by a hard and fast line. The acute type comes on suddenly. There is an initial dullness, followed by prostration and high fever. Heart action is more powerful and frequent than normal and is accompanied by venous regurgitation. The conjunctiva is injected, yellowish and deep red. Loss of nerve and muscle force becomes remarkable as the disease progresses. Emaciation is rapid. The temperature although variable is high (105°-107° F.) with frequent irregular remissions. Cardiac impact is conspicuously increased, but in a few cases the heart is weak. The pulse is rapid, soft and compressible. Edema of the dependant parts of the body and enlargements of the lymphnodes are seen, but not constant in its occurrence. A blood stained, watery fluid

frequently drops slowly from the nostrils. The fæces are often streaked with blood. Exertion provokes dyspnoea and palpitation of the heart. In cases lasting more than five or seven days there is a reduction in the hemoglobin, as well as in the corpuscles of the blood, but otherwise those changes appear to be insignificant. The number of leucocytes generally remains about normal.

The subacute type, spoken of by Mack, resembles the acute, but may be more insiduous in its onset. Its course ranges from a few weeks to several months and is marked by periods of apparent convalescence and subsequent exacerbations of the disease. A high, irregularly remittent, fever, a progressive anemia, a rapid emaciation, prostration, cardiac weakness and edema are characteristic features of this type of the disease. While the temperature may rise as high in this type as it does in the acute one, the periods of remission are longer. It rarely reaches the normal point and a greater irregularity is encountered. Locomotor, respiratory and cardiac difficulties become pronounced and are aggravated by exertion. Respiration may become labored. A venous pulse is noticed and the arterial pulse is soft and compressible. The lymphnodes may show enlargement. During the periods of high fever, the appetite may be interfered with, but otherwise it is keen enough.

In the chronic type the duration is longer. There are occasional slight febrile attacks, a weakened heart, jugular pulse and more or less loss of flesh and energy. Anemia is not marked until the later stages. In spite of temporary improvements a fatal issue is almost certain.

Whitehouse [56] recognizes several types of the disease. Those of the anemia type will run a very slow indefinite course. There will be a slight and gradual loss of flesh, with a staggering or swaying gait. The mucosa are pale. A constant and voracious appetite is invariable in this type and is accompanied by considerable thirst. There is generally a slight rise of temperature, but not over 103.5° F. The pulse runs from 50-70 beats per minute. As the disease

advances, polyuria develops. The various symptoms gradually increase, the duration of the disease covering from three weeks to six months, the animal wasting away to a perfect skeleton.

Another type is mentioned by Whitehouse as the edematous one. It closely resembles the anemic one and is very nearly as common. Dropsical swellings appear early in the disease, beginning with a small pad at the brisket and a slight enlargement of the extremity of the sheath. These edemata, may increase to a great size and the skin covering them may exude serum. The legs may swell, but do not often do so. Temperatures up to 104.5° F. are occasionally witnessed.

A different type is the one showing intermittent fever. In this type the initial temperature may suddenly run up to 107° F. After three days of illness the normal may again be reached. Twenty-one days later there is a repetition of the occurrence and after twenty-one days more another attack occurs. Whitehouse observed two such cases, one completely recovering after a third attack, and the other one succombing, while the third attack was in progress. This author also speaks of the bronchial type and calls it puzzling. His observations on this type are limited. The cases which he saw were only very slightly wanting in flesh and "appeared like animals left with a bronchial thickening after influenza." A fulminating type is mentioned by Whitehouse.

In a more recent Canadian publication, McGilvray [64] dwells upon the tendency to apply the name of "swamp-fever" rather promiscuously to a considerable variety of horse diseases. He recognizes three typical entities, which are more often covered by the name of swamp-fever and calls them: I. Swamp-fever; II. Typhomalarial fever; and, III. Low fever. No. I is characterized by its insiduous and chronic course, remittent fever, pronounced anemia, associated with a steady, progressive emaciation, in spite of a well maintained and frequently voracious appetite. No. II is an acute or subacute affection of horses characterized

by its sudden onset. There is an extremely high initial temperature, marked injection of the visible mucosæ, which later become icteric. There always is a marked disturbance of circulatory functions and extensive edema of dependant parts. A critical polyuria and diarrhea precede dissolution. No. III is distinguished by slight elevation of temperature, usually maintained, marked unthriftiness, lassitude and incapacity for work. The visible mucosæ are congested and frequently icteric. The appetite is capricious. Edema of the sheath is noticed. Polyuria is always more or less in evidence. The bowel function is irregular, the fæces frequently being coated with a slimy mucus.

Mohler [52] describes the disease as characterized by a progressive, pernicious anemia, by remittent fever, polyuria and gradual emaciation, in spite of a voracious appetite. This stage is followed closely by a staggering gait, swaying, uncertain gait, the hind legs being most affected. Weakness and tenderness in the loins are also mentioned. The pulse increases in rapidity and may run as high as 70 beats per minute, although weak, stringy and intermittent. The temperature may rise to 103° F. or higher, remaining high for several days and then dropping to rise again at irregular intervals. Toward the end of the disease, the temperature occasionally remains persistently high. Temporary improvements are noticed. Albumen appears in the urine in the advanced stages. Mohler occasionally observed a slow dripping of blood-tinged serum from the nostrils as a result of the oozing of thin blood from the mucosa.

Our own observations tend to show that fever is perhaps the most constant symptom mentioned in connection with the disease. It is always more or less irregularly remittent or intermittent in the field cases, the range of temperature commonly lying between 101° F. and 104° F. Towards the last it may have a tendency to be more constant, although this feature is not to be depended on as of constant occurrence.

In the experimental cases, it is the first of all symptoms and in quite a number of cases about the only one sufficiently pronounced to have a diagnostic meaning. In most of those cases it makes its appearance after periods of incubation varying from 6-21 days, according to the method of infection practiced. After intravenous injection, for example, the incubation period will be found to be shortened when the same virus is used. It will be somewhat longer after subcutaneous injection, while when the virus be given by the mouth, it will be still more prolonged. In general the period of incubation is longer when urine is used for infecting purposes than when blood is employed. It is, of course, not possible to fix the period of incubation definitely as variations in the virulence of blood and virus and the susceptibility of the experimental horse used are factors over which we have no control or which cannot be estimated. In the greater number of our cases we have found that when using fresh blood as a virus and injecting it subcutaneously in quantities of 60-120 c. c. we may expect the initial temperature rise in from 12 to 14 days. In such cases it sometimes happens, that the temperature rises to about 105.5° F. and then again drops down to normal for 12-24 hours, after which there is a steady increase in temperature, which reaches its height in from 2-4 days. The maximum temperature will usually amount to from 104°-107.5° F., but will not long remain so, as in a day or two, it will gradually go down either to normal or nearly so. This initial attack in most cases will last from four to ten days. In a few cases, the remission or intermission is of but short duration and is followed immediately by periods of more or less fever of indefinite duration. Such cases may run an acute course and carry an abnormal temperature until they die, while in other cases the fever may recede gradually after having lasted for several days. In most of our cases, the first months of infection witness a number of well defined fever exacerbations, separated by remissions and intermissions. At first they are apt to succeed in rather rapid succession, but

as time goes on they become divided over greater periods. In the further course of the disease, as induced by experimental infection, there will be noticed periodic rises often after rather markedly regular intervals (See chart of No. 636), varying from 30-60 days. In some of the cases this feature kept up for two years, although the fever periods occurred at greater intervals after the first year. In other cases the temperature phenomena did not reappear after a year, while the virulence of the blood persisted for much longer.

The pulse rate is often influenced by the temperature and to some extent the curves show some parallelism. In anemic field cases of long standing the pulse is soft and thready, conveying the idea of swishing through the palpating finger. In more vigorous experimental cases, the pulse is more voluminous and but for the increased frequency does not differ much from the normal. Towards the fatal termination, the pulse becomes smaller, weaker and at last imperceptible. In all cases, with the possible exception of the more vigorous experimental cases, during the fever intermissions, the frequency of the pulse ranges between 55 and 65 beats per minute and may even reach from 90-100, or even more towards the final issue.

During the latter stages of the disease are changes of cardiac origin noticed. The impact is increased in force and may be accompanied by a churning sound, when pericardial exudate be present. An anemic bruit has been heard in some old field cases but is not a constant feature.

No respiratory changes have been noticed, which could be attributed to specific changes. In one of our field cases there was a concurrent pneumonia, with the ordinary symptoms of respiratory distress. In some cases there, of course, are respiratory disturbances incidental to cardiac or general weakness. In one of our cases there was a foul smelling nasal discharge due to extensive necrosis of the septum nasi.

During the febrile attacks dullness is a noticeable feature. The animals will stand with heads down into the

manger or will hang back on their halters, as if wishing to support themselves in that way. In not a few of the cases, however, the fever exacerbations are not accompanied by any marked features and the rise of temperature and a little higher respiratory frequency are the only symptoms presented.

Under the conditions just mentioned, the appetite is usually reduced or entirely suspended. An idea prevails that in "swamp-fever" a normal or even an abnormally increased appetite is a constant feature. This is not so, as we have seen in many cases of the disease and especially during the febrile periods, that the appetite shows the same irregularities that it does in other febrile diseases. It is, however, true that in many of the chronic and anemic cases, a voracious appetite is a notable phenomenon and as such cases are probably the only ones usually diagnosed, we cannot wonder that an increased appetite is so often mentioned as something characteristic of the disease.

Weakness and lack of muscle tone is a prominent symptom in the anemic cases and even in the cases running the course without anemia, it is not infrequently a marked feature. The animals readily tire and blow, when exercised, they will knuckle over and stumble when moving and they present a well marked swaying, wabbling motion with the hind quarters, which seems to be the principal seat of muscular weakness.

Loss of flesh usually accompanies the febrile attacks and in quite a number it is progressive, the animals reaching a stage of extreme emaciation. Affected animals will frequently present a dull, staring coat, while the condition known as hide-bound usually prevails whenever emaciation is present. In other cases the state of nutrition improves during the non-febrile intervals, but when anemia becomes a feature, emaciation becomes a more or less constant phenomenon.

Edema of the dependant parts of the body, such as the lower thoracic and abdominal wall, the sheath, the limbs, is

a common feature. It is more commonly seen toward the latter stages of the anemic cases, when it is due to the blood changes, combined with cardiac and probably also renal insufficiency. We have also observed marked edema, however, in the acute experimental cases, in which anemia was not a feature and in which the blood count even ran higher, than what is usually considered normal.

Anemia was certainly a more or less marked symptom in all but one of the field cases which have thus far come to our attention. The visible mucosæ are pale, even blanched, and the blood count will reveal a sub-normal number of red cells, usually ranging between two and four millions, although in one of our field cases, the count amounted to less than a million. In the experimental cases, anemia is by no means a constant feature, in fact, it was only in a few cases that artificial infection expressed itself by a marked anemia and several of the cases went to a fatal termination, with a normal blood count or even with a number of erythrocytes considerably above normal. The hemoglobin percentage is often reduced and this reduction is frequently greater than the degree of anemia would indicate.

Not uncommonly the blood clots but slowly and occasionally separation of clot and serum is imperfect. In such instances, the cellular constituents of the blood have time to settle to the bottom of the vessel before the entire contents are changed into a rather soft coagulum. The serum frequently shows a high color, sometimes a greenish cast. Occasionally there is a tendency on the part of the erythrocytes to clump together, so that the thorough mixing preparatory to counting becomes either difficult or impossible.

The changes in the corpuscles are not very marked. The erythrocytes are somewhat fainter in the anemic cases and may in the same blood, show a more or less conspicuous variation in size. Crenation is not uncommon, but typical rouleau formation is not often seen. A slight poikilocytosis has been observed in one or two cases only, but in none of the cases

examined by this department were nucleated erythrocytes found. As far as our cases go, the blood always contains the specific virus of the disease, but a search for visible parasites which could possibly be associated with its causation always resulted negatively. In some of our cases bacteria were found in the blood, but there was no constancy as to the species nor were there indications that they were anything beyond mere accidental intruders.

The blood may be highly virulent in the absence of all other symptoms or phenomena. We know this virulence to exist for not less than thirty-five months in an experimentally infected case, and further investigations will probably reveal the fact that it is retained for a much longer period. We do not believe that in all of the cases of this character the blood remains permanently virulent as there are some indications that warrant the belief that after a long time its virulence becomes less and finally disappears. Our investigations, however, have not yet advanced far enough to permit of a definite statement in this regard.

Albuminuria is frequently seen in cases of "swamp-fever," both in the field and experiment cases. It is not a constant accompaniment of the disease; sometimes it will be in evidence in a given case, while a little later, it may have completely disappeared. At no time have we found the amounts of albumen, mentioned by European authors. In a few cases there was a sharp and well defined albumen reaction, but in the greater number, only traces were present. Only in one case were we able to find tube casts in the urine.

Polyuria is occasionally seen and especially towards the latter stages of the anemic field cases.

The chronic cases usually die from general exhaustion, supplemented by cardiac weakness, while in the more acute cases, the immediate cause of death is not explained by the lesions found after death.

In some of the latter types of cases, we found that death could be attributed to thrombosis of the pulmonary vessels,

while in others no anatomic changes explained the exitus lethalis. Here we probably had to deal with a true intoxication and subsequent interference with the more essential parenchyma.

When we carefully analyze the clinical features presented by our cases. we will be struck by the regular presence of anemia in the field cases and its rather uncommon occurrence in the cases in which the disease was experimentally transmitted. The blood of field cases never failed to induce infection into the experimental horses and from them it could be passed into other horses again with almost unbroken regularity and often with fatal results and yet in no case were the symptoms of the original field case fully duplicated.

Were it not for the fact that this was regularly the case in all experiments involving six field cases from different sources of origin, we might be warranted in believing that the original infection had not been transmitted, but that some other virus had accidentally interfered with our work. The repetition of our observations, and the findings during the autopsy, however, would preclude such a conclusion. It may be suspected that in our experimental cases certain contributory causes are not in operation. Such cases are well fed, housed and not exposed to the wear and tear incidental to hard farm work, while in our field cases the opposite is not uncommonly the case. We will not deny the influence of the factors mentioned, but the fact that many of our experimental cases, in spite of the complete absence of anemia, terminate fatally, seems to indicate that the virus introduced is capable of producing fatal disease without any contributory cause cooperating in the process.

In an endeavor to explain this peculiar finding another theory may be advanced, namely, that the anemic cases, are the only ones which are diagnosed as "swamp-fever" and that perhaps they only constitute a certain fraction of all swamp-fever cases. The clinical observations made on our non-anemic experimental cases, convince us that, had they

occurred under actual field conditions, they would either have escaped attention altogether or would not have been diagnosed as swamp-fever.

In just this type of cases, we would never suspect the disease, if we did not know that they were artificially infected, as the symptoms presented are so extremely indefinite as to most commonly preclude a clear cut diagnosis. We lean, therefore, more or less to the opinion that the anemia so commonly associated with the disease is by no means a constant occurrence and that we will have to reconcile ourselves to the idea of non-anemic swamp-fever cases, in other words that in only a certain number of the cases anemia is a feature. We have a similar instance in the case of carcinoma in man. There, anemic conditions under certain circumstances are considered sufficient to warrant the suspicion of cancer, in fact, cancer is frequently accompanied by anemia. Yet many cancer cases exist, continue and die without anemia being a feature.

Unfortunately, we have no reliable field data, covering this point, but we may mention the fact that often, when making inquiry regarding swamp-fever outbreaks, informers will tell us something like this: "Yes, we lost quite a few horses, but only a few died with swamp-fever, as the others that died were sick only a few days and it takes swamp-fever much longer to kill a horse."

We are in hopes to carry our experimental work into the field during the coming season and then this point will be more thoroughly looked into.

If the above opinion be correct, it seems that the diagnosis offers greater difficulties than was heretofore supposed. Most authors seem to regard the diagnosis of "swamp-fever" as something easily accomplished, and but little space is devoted to its discussion.

Köpke [8] mentions "Brust-seuche" (infectious pneumonia), febrile gastro-enteritis and pernicious anemia as of importance in the differential diagnosis, while Torrance [9]

regards the dragging gait, the good appetite, accompanying loss of flesh, anemia and the peculiar thrilling pulse as diagnostic evidence. Peters [26] does not consider the diagnosis to be very difficult during the later stages of the disease. He considers the anemia and progressive emaciation, combined with the good appetite, as good grounds for a diagnosis. Hempel [49] moving along more rational lines experiments with a view of determining the existence of the disease by the complement fixation method, but his results appear to indicate that this method cannot be relied upon to become an aiding factor in the diagnosis. Hutyra and Marek [69] take into consideration influenza catarrhalis, anthrax, enzootic spinal paralysis, piro-plasmosis, Sclerostomiasis and simple anemia when dealing with the differential diagnosis of the disease. To Mack [50] the correct diagnosis of swamp-fever does not appear to be without its problems, at least, he speaks of the difficulty of differentiating this disease from other conditions accompanied by anemia. In this differential diagnosis, he attaches some importance to the relative increase of lymphocytes and the corresponding decrease in the polynuclear cells of the blood. He also lays stress on the value of eosinophilia as an evidence of helminthiasis. The acceleration of respiratory and cardiac activity after exercise is also looked upon by Mack as a valuable aid in the diagnosis. Mohler [52], on the other hand, states that the diagnosis is not difficult and he regards the symptoms, usually met with in swamp-fever cases as sufficiently characteristic to warrant a diagnosis. Kinsley [63], when making the diagnosis is guided by the presence of a high temperature, anemic pulse, anemic cardiac murmur, jugular pulse, petechiæ on or a muddy appearance of the conjunctiva, relaxation of the sphincters, enlargement of the sub-maxillary lymphnodes and general depression. To this he adds that locality and history must be given due credit and asserts that "blood inoculation is the only positive method of diagnosis in this disease." According to a Prussian source [55] "infectious anemia" may be suspected when

there are observed: dullness, a bad state of nutrition, pâle and pinkish white mucosæ, reduction in the erythrocyte contents of the blood, increase in the pulse frequency (especially marked after exercise). To those phenomena there may be added, fever, subcutaneous edema and albuminuria. In one of the cases studied by Todd and Wolbach [75], the animal when first examined appeared healthy, the blood count amounting to 6,500,000 erythrocytes, which in the course of three months rose to 8,500,000 and the authors add that, were it not for the distinct rises of temperature which followed in the horse which was inoculated with the blood from the case, it would be permissible to question whether it were ever infected with swamp-fever. The same authors record a blood examination in a horse four months after inoculation and which was down and unable to rise and yet the count revealed 10,640,000 red corpuscles and 29,500 leucocytes per cubic m. m., while the hemoglobin amounted to 115 per cent.

If "swamp-fever" under natural conditions only occurred in the form of the anemic disease, which we had occasion to describe in a former paragraph, the problem of diagnosis would become simplified to a remarkable extent. But even then a clear cut definite diagnosis, such as is possible in so many of our infectious diseases, would often be impossible if we had to depend on objective symptoms alone. Accepting the experimental evidence of the existance of a non-anemic form of the disease, and especially with reference to what may be called non-clinical infection carriers, we find that the task of correctly diagnosing the disease is not only highly difficult, but more frequently even absolutely impossible with the means and knowledge now at our command. In fact, for so far as our observations go, we have come to recognize in the problem of the diagnosis, the most difficult phase in our investigations. It is true that for purposes of experiment, we have recourse to the inoculation of a healthy horse, but this method of diagnosis can only find a very limit-

ed application in actual every day practice. The absence of an absolutely reliable method of diagnosis, aside of offering an enormous obstacle, also places a great part of field and experimental data in the doubtful column. Every so often we learn of a certain line of treatment, for example, which proved to be beneficial in "swamp-fever" or even "cured" cases of the disease, but before accepting those statements at their face value we may well question, what evidence is there of an absolutely correct diagnosis and if the diagnosis were correct by what method was the cure proven?

Under the existing difficulty of diagnosis, a greater part of the evidence on swamp-fever becomes worthless and under these conditions we have come to accept only such evidence as can be supported by actual inoculation tests.

In spite of unreliable objective symptoms, a diagnosis is however, often possible, especially in the anemic type of the disease. Aside of the ascertaining of the various symptoms mentioned above, an inquiry into the circumstances surrounding or preceding the case under observation, is imperative if we wish to be guided toward correct conclusions. An anemic fever case, with occasional albuminuria, edema, a swaying gait is in itself sufficient to arouse suspicion, but when this evidence is supplemented by a history of several, previous cases of the same nature and this all in a locality reported to be a swamp-fever center, it becomes quite safe to make a swamp-fever diagnosis. Such supplementary evidence, may and would also aid in arriving at a plausible conclusion in the face of indefinite non-anemic illness, marked by remittent or intermittent fever, even if an unimpeachable diagnosis cannot be obtained. The question of picking out from an infected herd, the non-clinical infection carriers, however, important, is as yet very difficult. Unless we resort to inoculation experiments, we could only offer one suggestion and that is to record the daily temperature for long consecutive periods. Such cases, the great majority of them at least, will sooner or later show their typic febrile exacerbations and this at least would be an indication of some

practical value. However. it must not be forgotten that in some of the cases of this type, no fever reaction occurs and that if it does occur, that we are not in position to definitely attribute it to swamp-fever infection. Here possibly, the demonstration of albuminuria may help out, although this condition could very well occur in other infections.

After carefully considering our evidence in the light of our present knowledge, we must conclude, that with the exception of the diagnosis of the anemic cases, with a history of infection, we are not able to promptly recognize the disease. We maintain, therefore, that in the matter of diagnosis, investigators have as yet to solve, perhaps the principal problem of the disease.

Abderhalden and Frei [67] and Abderhalden and Buchal [74] report a series of experiments, which tend to show that the blood serum of horses infected with pernicious anemia exerts a marked inhibitory action upon the hemolysis induced by saponin. As the reaction was not studied in case of other febrile diseases, our judgment of the practical value of this reaction must as yet be suspended, but at any rate the findings of the authors mentioned should stimulate further investigation.

In view of the difficulty of correctly diagnosing "swamp-fever" in all its forms, the data on the prognosis must also be accepted with a considerable amount of caution and it seems probable that the information available principally pertains to the anemic cases. Of the sixteen cases observed by Köpke [8] nine terminated fatally and of those one colt died on the fourth day of sickness. In the cases running a favorable course, the temperature gradually receded to normal in from six to eight days, and Köpke states that they made a complete recovery. Torrance [9] says that the duration of the disease extends over two or three months, but that in some more aggravated forms, the animals die in from two to three weeks. Torrance considers the prognosis as

being very unfavorable and places the mortality figure between 50 and 100 per cent. In the opinion of this author, a small part of the cases recover when treatment is begun early and when the erythrocyte destruction has not exceeded a certain limit. In the report of Brimhall, Wesbrook and Bracken [10] the unfavorable prognosis and the futility of medicinal treatment are dwelled upon. They estimate the mortality of the cases at 80 per cent. In Rutherford's [11] experience recoveries are very rare. He says that apparent recoveries are sometimes noted, but a sudden relapse is not uncommon in such cases. Jarmatz [14] places the mortality at about 10-15 per cent if suitable treatment is begun soon after recognition. Ostertag [35] states that recovery from the typical disease is exceptional and requires a long time. Hutyra and Marek [69] inform us that recovery seems to be a rare exception and that if it takes place at all, it may only be apparent. Mack [50] places the mortality figure as probably well above 90 per cent of the cases. In Mohler's [52] description of the disease, the mortality is rated at 75 per cent or even higher. He states that recovery takes place only when treatment is begun early or when the animal has a long convalscent period, but follows this up with the statement that the treatment of the disease "has so far been far from satisfactory." Kinsley [63] advises a guarded prognosis, although he gives the opinion that if the veterinary directions were followed the mortality would not be over 25 per cent.

It is, of course, impossible to construct mortality statistics from the cases, field and experimental, that came under observation of the writers, as in a number of cases the cause of death was open to question while in not a few instances, the animals, although infected, were not permitted to live long enough for the disease to run a natural course.

The following table gives a review of the fate of our cases:

	Field cases	Exp. cases	Remarks
Still alive	1	6	
Died or killed in moribund state	5	24	Among those there are five of which the cause of death is doubtful
Infected but killed in fair condition	....	13	
Cases resistant to infection	....	4	One of those was destroyed on account of bone fracture

In a disease in which the diagnosis is manifestly beset by so many difficulties, most data on prognosis can only be accepted with suspicion as to their accuracy. In the type of the disease, that is commonly diagnosed as "swamp-fever," we are safe in expecting an unfavorable termination, and in placing the mortality percentage as ranging between 90 and 100 per cent. Recoveries have often been reported but such reports mean but little if they are not substantiated by positive proof that the blood of such cases is no longer virulent. In a disease of infection and evidently transmissable, recovery should not only be meant to include restoration to health, but also that the case under consideration has ceased to be dangerous as a virus carrier.

There are indications, that recoveries may be more common among the non-anemic forms of the disease, but it also seems that recovery only takes place after a very long period, marked by indefinite febrile attacks and their accompaniments. As such cases, however, are actively infective during all this long time, it seems that a fatal termination after a brief illness, would be more economic than the actual recovery from the disease. It should not be overlooked, however, that a number of the non-anemic cases die early in this disease (See chart of No. 934), but we have not a sufficient amount of data at our disposal to warrant the naming of a definite death rate for this type of cases or for the disease as a whole.

As a general rule the presence of anemia, edema, emaciation, weakness, albuminuria and polyuria must be looked upon as bad omens. In the acute non-anemic cases, continued fever even if more or less remittent in type, or fever exacerbations following one another in rapid succession point toward a short duration and fatal termination. In those cases also we must look with apprehension upon a continued albuminuria and the presence of edematous swellings of the dependant parts of the body.

On the whole, the disease in all its forms will justify a rather unfavorable prognosis.

It is not a surprising feature that in so fatal and so damaging a disease as "swamp-fever" or as some authors prefer to call it "infectious anemia" numerous attempts have been made to save the animals affected by various methods of treatment. Köpke [8] directed his efforts so as to secure a proper diet and disinfection of the intestinal canal, while aside of this he aimed to reduce the fever and to sustain the heart functions. Torrance [9] tried the use of quinine, arsenic, iron, mercury, Crédé's silver, various potassium salts, without discovering any line of treatment which was uniformly successful. According to Rutherford [11], the long continued use of arsenic, as recommended in surra by Lingard in India, combined with mineral and vegetable tonics and a change to high, dry grounds has perhaps been more satisfactory than any treatment yet tried. In a later report by Torrance [12] mention is made of therapeutic experiments with salol and that recovery followed in one of the cases, while the other case was lost by accident. From a subsequent report [15] it would appear that the recovery mentioned was only an apparent one.

Jarmatz [14] obtained the best results with white arsenic in quantities of 15 grains per day, supplementing this treatment by a highly nutritious diet. He mentions claret as to be of service also.

Ries [27] uses the following treatment in his "infectious

anemia'' cases; viz., assimilable food, hygienic care and surroundings, stimulants, febrifuges, tonics, blood builders. When fever is present 15 grammes of quinine sulphate are given at one dose, while at each meal a bottle of claret is administered and a few liters of cold water are injected per rectum about three or four times per day. Against the albuminuria, which is a usual accompaniment, Ries uses a milk diet. In the morning ten liters of sweet cold milk, are given and immediately after some crushed or whole oats with a little bran. To this starchy ration the author adds, morning and evening, a spoonful of cooking salt and the same quantity of phosphorated oil (1:300). At evening the patient is watered, with water containing iron prepared by permitting water acidulated with 30-40 grammes of hydrochloric acid to act on rusty iron. When the disease reached the stage where the test tube revealed a clot which only was one-fifth of the total quantity, two patients, showing voluminous sub-ventral edema received morning and evening eight liters of physiologic salt solution until from 80-100 liters were thus given, but notwithstanding a slight improvement, after the first injections at least, death was not prevented.

Ostertag [35] relates an experiment with atoxyl as a therapeutic agent. The subcutaneous administration of doses of 10 and 15 c. c. of a ten per cent solution of atoxyl was followed by a marked improvement in the clinical appearance of two cases, although notwithstanding this apparent improvement, the hemoglobin percentage and red blood cell count showed a reduction.

Friedrich [44] reports that iron and arsenic preparations were given with unsatisfactory results. Apparently better results were obtained by the use of bicarbonate of potash, but Friedrich expresses the fear that the improvement obtained is only a temporary one, in view of the remittent character of the chronic form of the disease.

In a later publication Ries [47] mentions a trial with atoxyl and concludes that it is both harmless and useless.

His best results were obtained with infusion of salt solution.

A strenuous therapeutic activity is displayed by Acres [48] who used, either in combination or separate, liquor cresol, strychnine, potassium-chlorate, anti-febrine, ferric-sulphate and nux vomica. He also tried quinine sulphate, potassium iodide, resublimed iodine, arsenic, hydrochloric acid and "other drugs," but obtained the best results from the anti-febrine treatment. He qualifies this statement, however, by saying: "The animal, however, is generally subject to several relapses, which none of the drugs above mentioned seem to prevent." Acres winds up by trying antistreptococcic serum and states that his results were so encouraging that "it would be worth while giving this serum a thorough test."

Hempel [49] also experimented with atoxyl as a therapeutic agent. It was given in doses ranging from 0.5 G.-4 G., by gradual increase. Apparently the atoxyl treatment resulted in a marked improvement of the cases, although the blood of a horse thus treated was still infectious after it had received 26 G. in the course of about six weeks.

Hutyra and Marek [69] state that thus far no medicinal treatment of promise has been found, but make mention of the fact that certain arsenic preparations, at least in certain cases, seem to have a favorable influence.

Kinsley [63] gives a first place to absolute rest in the stable because "sunshine has a decided injurious effect upon the diseased animals." Cold baths and enemata to reduce the temperature in the acute type, while stimulants and the use of arsenic are relied upon for the rest. He states that this line of treatment continued from 4-6 weeks apparently completely destroys the causative agent of the disease, at least, the animals recover.

Mohler [66] seems to approve of the use of certain drugs but he regards the results treating the disease as far from satisfactory.

Van Es [68] thinks that in a therapeutic effort, iron and

arsenic, perhaps are best indicated, but that even with them no marked results can be anticipated.

Francis and Marsteller [81] made some therapeutic experiments with quinine, trypan-blue, trypan-red and atoxyl. The results of the latter three drugs were either doubtful or negative, but the authors speak of some encouragement from the use of quinine. It may, however, be mentioned that the case in which quinine was used, was under observation only for a short period afterward, and that the virulence of its blood was not tested after the treatment.

Whitehouse [83] used trypan-blue, giving only one injection of three grains dissolved in 200 c. c. of sterile cold water or physiologic solution, and expresses the opinion that in one of his cases recovery was due to the trypan-blue treatment. The results of the treatment apparently were not verified by experimental evidence.

From the above it would seem that the present status of swamp-fever therapy is as yet deplorably inefficient, and that in the way of treatment, we have nothing that promises to be specifically useful. In the absence of such a treatment, we will have to confine ourselves to meeting the symptoms as they arise, if we consider it worth while to do anything at all. Antipyretic treatment, medicinal or otherwise may be employed during the febrile periods, while in the anemic cases the use of the arsenic and iron may be tried. At one time it appeared that we obtained good results by giving six grains of arsenic, mixed with one drachm of the carbonate of iron on the feed morning and evening, and continued for a month at a time, but as neither our diagnosis nor the results of our treatment were verified by means of inoculation experiments, this evidence will have to be rejected as insufficient. As long as the horse owners are bound to demand medicine, to be swallowed by their sick horses, the arsenic-iron combination can perhaps not readily be improved upon, at least, it will in many cases prevent the use of irrational or freak remedies.

Whatever treatment be undertaken, it should be supplemented by the use of proper food and water and by placing the patient in hygienic surroundings.

A beginning has been made with some therapeutic experiments, which we hope to continue as the proper material for it becomes available.

The first trials concerned a preparation, most widely known as trypan-blue and which had gained some reputation abroad as being of use in certain diseases of infection caused by protozoa. The firm of Meister Lucius und Brüning, Hoechst A. M., Germany, was so kind as to place a liberal quantity of trypan-blue at our disposal and the following experiments were tried with it.

Experimental horse No. 855, shortly after its initial fever was given an intravenous injection of three grammes trypan-blue in 250 c. c. physiologic salt solution and the injection repeated every other day until six injections in all were given. There was apparently no constitutional disturbances caused by the injections. The mucosa became of a dense, ashy blue color, after the first injection and this blue was still apparent some six weeks after the last injection. During the three and one-half months which the animal was permitted to live, there were no fever exacerbations, although on a few occasions the temperature ranged between 101° F., and 102° F. When 48 hours after the third trypan-blue injection the blood of this case was drawn it still proved to be virulent.

A subcutaneous injection, of 60 c. c. of this blood made after the last dose of typan-blue into experimental horse 873, caused this animal to become infected, showing the initial fever between the 10th and 21st days after inoculation. Immediately after the first febrile period and during part of the second one, this animal received seven intravenous injections of 3 grammes of trypan-blue in 250 c. c. of physiologic salt solution, and sustained the injections very well. The mucosa and the urine became quite blue but otherwise, there were no ill effects attributed to the drug. Up-

to-date this animal's temperature has not been above 102.5° F., although the animal, while not anemic, looked unthrifty most of the time. When this horse's blood, however, was fed to experimental horse No. 903, it again produced positive infection.

While we hope to resume the experiments with trypan-blue, it would appear from the above experiments that it cannot be relied upon to destroy swamp-fever virus in vivo.

The one experiment, systematically made with atoxyl, resulted similarly.

Again using experimental horse No. 873, the following subcutaneous injections with a 10 per cent solution of atoxyl in physiologic salt solution were made:

Date	Amount	Date	Amount
9-23-10	10 c. c.	10- 8-10	40 c. c.
9-24-10	10 c. c.	10- 9-10	40 c. c.
9-25-10	10 c. c.	10-10-10	50 c. c.
9-26-10	15 c. c.	10-11-10	40 c. c.
9-27-10	15 c. c.	10-12-10	40 c. c.
9-28-10	15 c. c.	10-13-10	30 c. c.
9-29-10	20 c. c.	10-14-10	30 c. c.
9-30-10	20 c. c.	10-15-10	25 c. c.
10- 1-10	20 c. c.	10-16-10	25 c. c.
10- 2-10	25 c. c.	10-17-10	20 c. c.
10- 3-10	25 c. c.	10-18-10	20 c. c.
10- 4-10	25 c. c.	10-19-10	15 c. c.
10- 5-10	30 c. c.	10-20-10	15 c. c.
10- 6-10	30 c. c.	10-21-10	10 c. c.
10- 7-10	30 c. c.	10-22-10	10 c. c.

After the tenth injection there was a slight attack of colic but otherwise nothing out of the ordinary was observed. During the injection period the animal lost flesh a little, but no evidence that the atoxyl had anything to do with it could be detected.

During the month of treatment this animal thus received 71 grammes of atoxyl and yet, when a week after the last dose, this animal's blood was drawn and injected into experi-

mental horse No. 939, it produced disease after an incubation of ten days.

An as yet incomplete experiment with the use of quinine may be reported at this time, because the results thus far obtained already, indicate more or less what is to be expected when the returns will be complete.

Experimental horse No. 638 was given a series of intravenous injections of the hydrochloride of quinine, dissolved in physiologic salt solution, according to the following schedule:

6-22	1.0	500	.....
6-24	1.5	500	.....
6-26	1.5	500	.....
6-28	2.0	500	.....
6-30	2.0	650	.....
7- 2	2.5	650	.....
7- 4	3.0	650	.....
7- 6	3.5	650	.....
7- 8	4.0	650	.....
7-10	5.0	750	Slight tremors of Tensor fasciae latae.
7-12	7.0	900	Tremors well marked.
7-14	8.0	900	Tremors still more marked. An increase in pulse frequency of 5 beats per minute.
7-17	9.0	1000	Tremors involved more muscular regions of hind quarters.
7-19	10.0	1200	Tremors same as before. Increase in pulse rate 15 p. m.
7-21	11.0	1350	Subject restless; tremors extending to abdominal muscles. Pulse increase 20 p. m.
7-24	12.0	1500	Tremors general. Subject sways while moving. Pulse increase 30 p. m.
7-26	13.0	1750	Symptoms of 7-24-11 duplicate but more intense. Pulse increase 45 p. m.
7-28	14.0	2000	Muscular phenomena same as before.
7-31	15.0	2250	Marked muscular incoordination. Subject staggered. Pulse increase 65 p. m.

Between the tenth and fifteenth day after the last injection No. 638 sustained a typical fever exacerbation, while experimental horse No. 1068, which was injected with a quantity of serum drawn from No. 638, immediately before the quinine injection of 7-17-11 developed fever 24 days after inoculation.

Another experimental horse was injected with blood of No. 638 one week after the latter had received its last quinine injection, but as the usual incubation period has not yet

terminated the evidence expected from this case cannot at this time be incorporated in our report.

The indications at this time thus, very strongly point that but little is to be expected from the use of medicines in the management of this disease. Like in the case of most diseases of infection, we will have to direct our attention more particularly to possible means of prevention.

Torrance [15] advised farmers through the agricultural press to avoid pasturing their horses and to water them only from wells and this advice has had a good effect in diminishing the number of cases and reducing the loss.

Carré and Vallée [19] formulated the following rules of prevention: 1. Isolate or still better destroy the sick animals, or at least, gather up and disinfect their liquid and solid ejecta. 2. Do not offer to drink, in infected regions, to healthy subjects anything but spring water or water which was previously boiled. 3. Never to place a horse coming from a suspected district, among healthy animals, without being certain beforehand that the urine does not contain albumen.

Ries [27] who, as will be remembered suspects "bots" of having an etiologic relationship to the disease, recommends thorough grooming and in particular the removal of the eggs of Gastrophilus and the early treatment with bisulphide of carbon on farms where the disease breaks out or where there is reason to suspect the presence of bots in the stomach.

In the opinion of Ostertag [35] it is primarily necessary in order to guard against the introduction of the disease, to exercise increased care in the purchase of new horses. Furthermore, new horses should be kept completely separated from the other occupants of a stable and all, opportunities of infection by means of contaminating the water or food supply should be carefully eliminated. He also insists on a water supply of unquestionable purity. Those preventative measures are recommended to be kept up for three

months in view of the insiduous nature of the disease. When the disease appears in a stable, the affected animal is to be immediately segregated and the stable is to subjected to a thorough disinfection under veterinary supervision. Impervious stable floors are regarded as imperative in thorough disinfection. Manure from infected animals will be rendered innoxious by keeping it packed for one month in quantities of at least one cubic meter (1 meter equals 40 inches). Ostertag further recommends that the attention of horse owners be directed to this disease and that they be cautioned to be careful in making new purchases.

Béghin [22] recommends segregation of cases, disinfection of the stable and harness, as well as general hygienic measures.

The prophylaxis outlined by Hutyra and Marek [69] includes the prevention of the introduction of any horses, which are in a poor, anemic and weak condition, and of which the cardiac frequency increases markedly on slight provocation and which have albuminuria. In infected districts new purchases should be segregated for three months. For infected stables, isolation of the diseased horses and thorough stable disinfection are recommended.

Mohler [52] proposes segregation of healthy from sick horses, and thorough disinfection of premises and utensils.

Van Es [68] outlines a plan of prevention and mentions separation of healthy and diseased animals, and where this is not possible, the destruction of the diseased ones. The elimination of animals suspected of being "infection carriers" is also spoken of as well as thorough stable disinfection. On the suspicion that biting insects may be instrumental in the transmission, he also recommends that horses be protected against pests of this type. In conclusion, he recommends that "swamp-fever" be included among the quarantinable diseases.

As a transmissible disease, swamp-fever, must in the first place be dealt with like we deal with any other in-

fectious disease. If we are to confine this disease to its present centers of infection, it will be necessary to exercise some supervision over the horse traffic in and from the infected districts. In fact, it would seem wise to suspend all movements of horses from an infected region, at least, for a certain time of the year, say from July to December, as evidently most of the actual transmissions take place during that time.

Animals showing such symptoms, as would warrant the suspicion of swamp-fever, certainly should not be moved from place to place, even after they have made apparent recovery, because of the now well established fact that such cases will remain actively infective for a long period.

Especially important is care on the part of farmers, having healthy stock, when introducing recently purchased horses into the stable. Owners of non-infected stables within the sections where the disease is prevalent, would do better to make their purchases outside of the district than within it, if they are in need of new horse stock.

Complete segregation of animals having the disease or suspected of having it must be carried out rigidly and the best way to do this is to leave the infected animals where they are and remove the horses, which are still in good health. Better still would it be to destroy the actual cases. In the great majority of instances such animals die anyway and the longer they are kept the more liable will they be to infect other stock.

When once the disease has made its appearance among a certain lot of horses and the apparent cases of the disease eliminated by destruction or otherwise, the possibility of infection still lurking about in the body of some horse which does not show symptoms becomes a serious problem and here we feel most acutely the imperfection of our knowledge of this disease. The results of our own investigations, as well as those of Vallée and Carré [17], Ostertag [35], and Francis and Marsteller [42] clearly indicate that such cases will have to be reckoned with. As yet we have no clear

cut practical method by which such "infection carriers" or rather "infection distributors" may be detected. The best we can do under the circumstances is to keep track of the body temperature of all the horses in the stable, on the principle, that many of those so-called occult cases will from time to time show the typical transitory fever mentioned in another paragraph. In addition, we may examine the urine from time to time for the presence of albumen. Cases showing the characteristic temperature rise or albuminuria must be isolated on the ground of presenting symptoms, warranting the suspicion of swamp-fever.

Under all circumstances connected with real or suspected cases of the disease, thorough stable disinfection must be rigidly carried out. This must not be done in a perfunctory manner because much may depend on it.

In infected districts, damp, swampy or marshy pastures should either be avoided for horse stock or thoroughly drained. It is here where probably most of our infections occur, through the contamination of the foliage and water pools by the urine of swamp-fever cases and "infection carriers." It is also advisable to prevent horses from using the water found in ditches or pools of such pastures and instead the water supply should be derived from wells, protected against the contamination by surface drainage or seepage. The drinking troughs in which the water is offered should be well above the ground, so as to reduce the danger of contamination with body ejecta to a minimum. At the same time, the formation of small pools around troughs should be prevented, by providing a sufficient drain for the waste water. Undrained pastures which are unfit for horse stock may be safely used for other animals, for so far at least as "swamp-fever" is concerned. While probably most of our cases originate from pasture infection, there is plenty of evidence to show that stable infection also takes place and with that possibility in view, the stable should receive considerable attention in a hygienic way.

Remembering that we have absolute proof that the cause

of the disease leaves the body of the infected animal by means of the urine, we can readily see how this substance may find its way into the well if it be sunk immediately under the stable. This is a practice prevalent on many farms and constitutes a source of great danger in this, as well as in many other diseases.

There is really but one condition under which a stable or barn-well can be permitted and that is when the stable is provided with a good concrete floor and when stable drainage is conveyed away from the building by an adequate arrangement.

We wish to call the attention of the farmers of fever infected districts, especially to these facts, as we feel certain that on many farms the disease can be brought to a standstill by paying attention to them. The common wooden stable floor, with its space full of inequity under it, explains the presence of many of our most stubborn diseases.

The constant presence of the specific virus in the blood and the well known part played by biting insects in the dissemination of other diseases seem to warrant us to include the protection of our horses against those pests and the eradication of them among the preventative measures.

It is but natural, that when dealing with an infectious disease, some attention should have been paid to the possibility of immunity as a factor including a promise of practical application. Owing to a rather recent solution of the etiology of the disease, nothing definite on swamp-fever has been thus far published.

Carré and Vallée [23] make mention of the fact that certain apparently recovered cases sustain large doses of virulent material without experiencing any grave disturbances, while they state that a beginning was made by them on some immunity experiments, which are to be made the subject of a future publication. Hempel [49], however, after experimenting in order to decide if horses which had apparently recovered from the disease had acquired an active

immunity or not, found that such was not the case and that such animals seemed to be still susceptible to a second infection. Mohler [23] speaks of investigations being in progress with a view of producing a serum or vaccine for immunizing purposes. Melvin [70] concludes that natural immunity against swamp-fever does exist in some horses and that this immunity may be increased by repeated injections of virulent serum from which contaminations have been removed or by means of defibrinated blood.

Our own data on the question of immunity in "swamp-fever" are very scant. There are some indications that some of the experimental horses, which may have become mere non-clinical infection carriers or which perhaps have entirely recovered, are able to withstand large doses of virulent blood with impunity but our researches in that direction have scarcely advanced to the point, where it would be prudent to even think of conclusions. However, as a matter of illustration, the career of some of our cases may here be briefly summarized.

Experimental horse No. 637 was injected with a quantity of blood from field case No. 642 on August 11, 1908. As a result the animal sickened showing several marked febrile reactions between that and the latter part of November following. From then on to May 1909, the temperature remained practically normal. On March 20, April 13, May 14, May 30, and June 15, the animal received injections of virulent blood, from different animals, which were increased from 20 c. c. to 1100 c. c. After May the general run of temperature became a little higher but no sharp exacerbation took place until October 25, 1909, when the temperature rose to 103° F., and although no further injections were made for the time being, there were several distinct febrile attacks between that date and October 4, 1910, when a large quantity of virulent blood was injected. This was followed by an indefinite slight transitory reaction on October 12 and November 15, while a subsequent injection of a large quantity

of virulent field blood on December 10, 1910, provoked sharp reactions on December 17, 1910, and February 2, 1911.

Encouraged by the fact that blood from No. 637 on June 15, 1909 failed to produce disease, the writers were induced to test its possible immunizing properties. This was done by the simultaneous injection of the blood of No. 637 and that of some other animal known to be virulent.

Acting on this plan experimental horse No. 729 was on July 13, 1909 injected with 50 c. c. of virulent blood from No. 638 and 500 c. c. of blood from No. 637. The horse was found to be dead on the following morning. Experimental horse No. 788 was used for a similar simultaneous injection on July 15, 1909, and succombed after a febrile period of eight days.

The experiment repeated on experimental horse No. 804 resulted fatally in five days, while experimental horse No. 811 lived only three days after injection.

Repeating the experiment in experimental horse No. 816 the results were again fatal in 25 days. In this case a marked anemia (2,500,000 red cells,) was observed two days before death.

From those observations it was evident that the combination of the blood of Nos. 637 and 638 had to be discarded as a base for immunity experiments.

It should, however, be observed that the experiment repeated on experimental horse No. 859 about one year later did not provoke any visible results. The horse experimented on had shown a high relative or perhaps absolute immunity to previous injections, which may in a measure account for his surviving.

Experimental horse No. 638 was inoculated with a diluted virus, passed through a Pasteur Chamberland filter on 10-7-08, and showed several febrile periods, the curves showing their apices on the following dates: 10-18-08, 10-25-08, 12-10-08, 1-16-09, 3-4-09, 5-14-09, 10-18-09. Since that time the animal has shown only twice a very transitory rise of temperature, at no time exceeding 102.5° F. Be-

tween fever attacks and even after the last marked rise of 10-18-09, the animal has been a picture of health and vigor. Although No. 618 showed severe illness and great depression during several of its fever attacks, there never was the least evidence of anemia; in fact, the blood count always showed a higher figure than what is usually considered to be the normal standard for the horse.

However, in spite of what from a clinical standpoint may be considered a complete recovery, this animal's blood was still virulent on 5-20-10 to experimental horse No. 879, although the incubation period, longer than usual, (22 days) may perhaps be considered as an indication of attenuation.

This animal was injected with virulent blood according to the following table:

Date	Blood	C. C.
7-15-10	636	60
8-15-10	873	120
9-20-10	921	240
11- 1-10	919	550

The last injection was followed by a marked rise of temperature beginning 24 days after the injection.

The blood of No. 638 was still virulent on 5-3-11.

Among our experimental horses we found three animals which were naturally resistant to the infection.

Summarizing the results of our investigations, the writers feel warranted in submitting the following conclusions:

I. Swamp-fever is a disease of infection, transmissible by subcutaneous and intravenous injection and by ingestion through the alimentary canal.

II. The virus producing the disease is contained in the blood and urine of affected animals, but it is absent from the fæces.

III. The virus has thus far been demonstrated only in an ultra-microscopic form.

IV. The virus is resistant to the severe freezing weather of our more northern climates.

V. While not denying the possible transmission of the disease to healthy animals by means of insects and parasites, animals contract the disease naturally by the ingestion of food and water, contaminated by the urine of an infected horse.

VI. The disease is essentially a septicemia, anatomically marked by sub-serous and sub-endocardial hemorrhages in the more acute forms, by occasional involvement of the lymphnodes and spleen, by degenerative changes in the parenchyma of heart, liver, and kidneys, and probably also by certain alterations in the bone-marrow of the long bones of the limbs.

VII. The chief and most constant manifestations of the disease are fever and albuminuria. The former is remittent or intermittent, not uncommonly at more or less regular intervals, while the latter is transitory and frequently synchronous with the febrile exacerbations.

VIII. Many cases of swamp-fever terminate fatally without a marked reduction in the red blood cells, a fact denying the popular conception of "swamp-fever" being primarily an anemia.

IX. The blood of an animal may remain virulent for as long as thirty-five months after the initial infection, without the infected horse manifesting any clinical evidence of the fact.

X. Such non-clinical infection carriers probably play an important part in the establishment of more or less permanent centers of infection.

XI. Both trypan-blue and atoxyl are worthless in the treatment of the disease.

XII. In the light of our present knowledge we have to depend upon such prophylactic measures as the destruction of diseased animals, segregation of suspects, care in introducing new horses into the stable, the safe-guarding of food and water supply from urine contamination, pasture drainage and stable disinfection.

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EXPLANATION AND KEY TO PLATES IV, V, AND VI.

Plates IV, V, and VI indicate the various transmissions swamp-fever infection in the course of the research work done at the Veterinary Department of the North Dakota Agricultural College. The black figures indicate cases in which infection was positively transmitted, the white ones, the cases in which transmission failed and the pinto horses, in which results of infection were of a doubtful nature.

No. 562. A field case from the vicinity of Harwood, N. D.

No. 577. Experimental horse infected by means of intravenous injection of 8 c. c. of blood from No. 562.

No. 635. Experimental horse infected by means of a subcutaneous injection of 100 c. c. of blood from No. 640.

No. 636. Experimental horse infected by means of subcutaneous injection of 100 c. c. of blood from No. 635. Is still alive 35 months after injection.

No. 637. An experimental horse infected by means of a subcutaneous injection of 100 c. c. of blood from No. 642.

No. 638. An experimental horse infected by means of an intravenous injection of a filtered mixture of 100 c. c. of serum of No. 639 and 700 c. c. of physiologic salt solution.

No. 639. An experimental horse infected by means of a subcutaneous injection of 100 c. c. of blood from No. 637.

No. 640. A field case from the vicinity of Christine, N. D.

No. 642. A field case from the vicinity of Fargo, N. D.

No. 723. An experimental horse infected by means of an intravenous injection of a filtered mixture containing 100 c. c. of serum of No. 638, and 700 c. c. of physiologic salt solution.

No. 727. An experimental horse resisting infection with 500 c. c. of blood from No. 638 given subcutaneously.

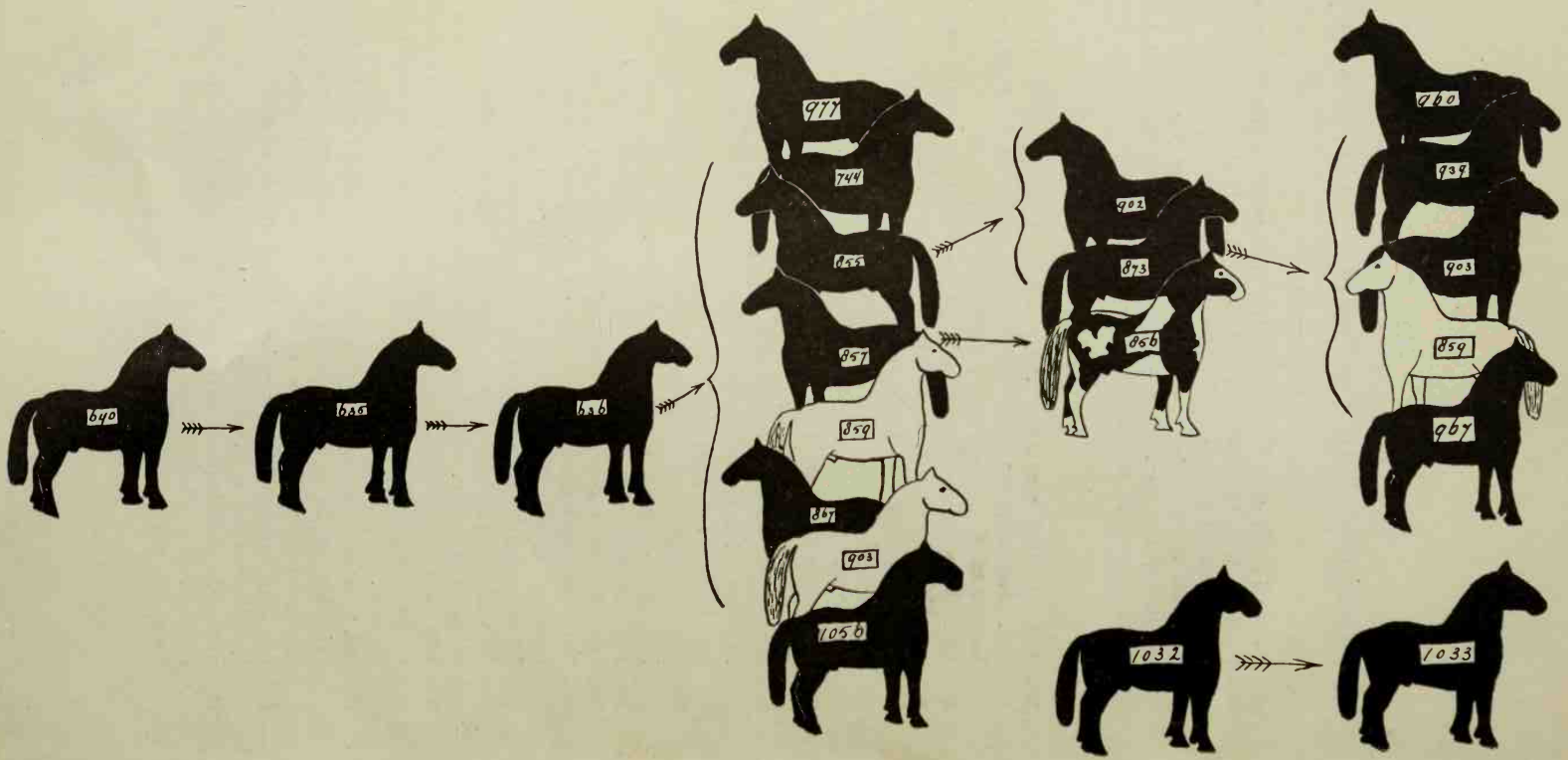
No. 729. An experimental horse, suddenly dying after an injection of 50 c. c. of blood from No. 638 and 500 c. c. of blood from No. 637.

No. 743. An experimental horse, infected by means of a subcutaneous injection of 50 c. c. of blood of No. 638 and after having resisted a similar injection of 130 c. c. of blood from No. 637.

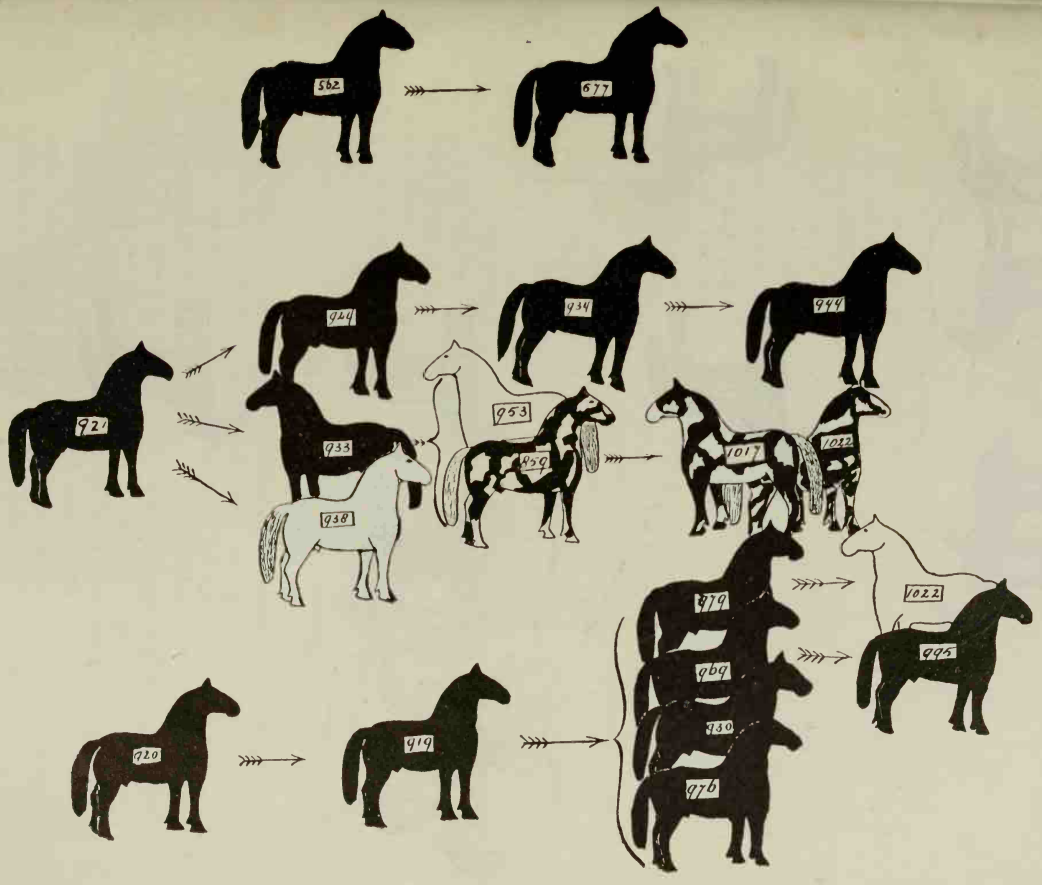
No. 744. An experimental horse, infected by means of a subcutaneous injection of 50 c. c. of blood serum from No. 636.

No. 788. An experimental horse, suddenly dying after an injection of 50 c. c. of blood serum from No. 638 and 500 c. c. of blood from No. 637.

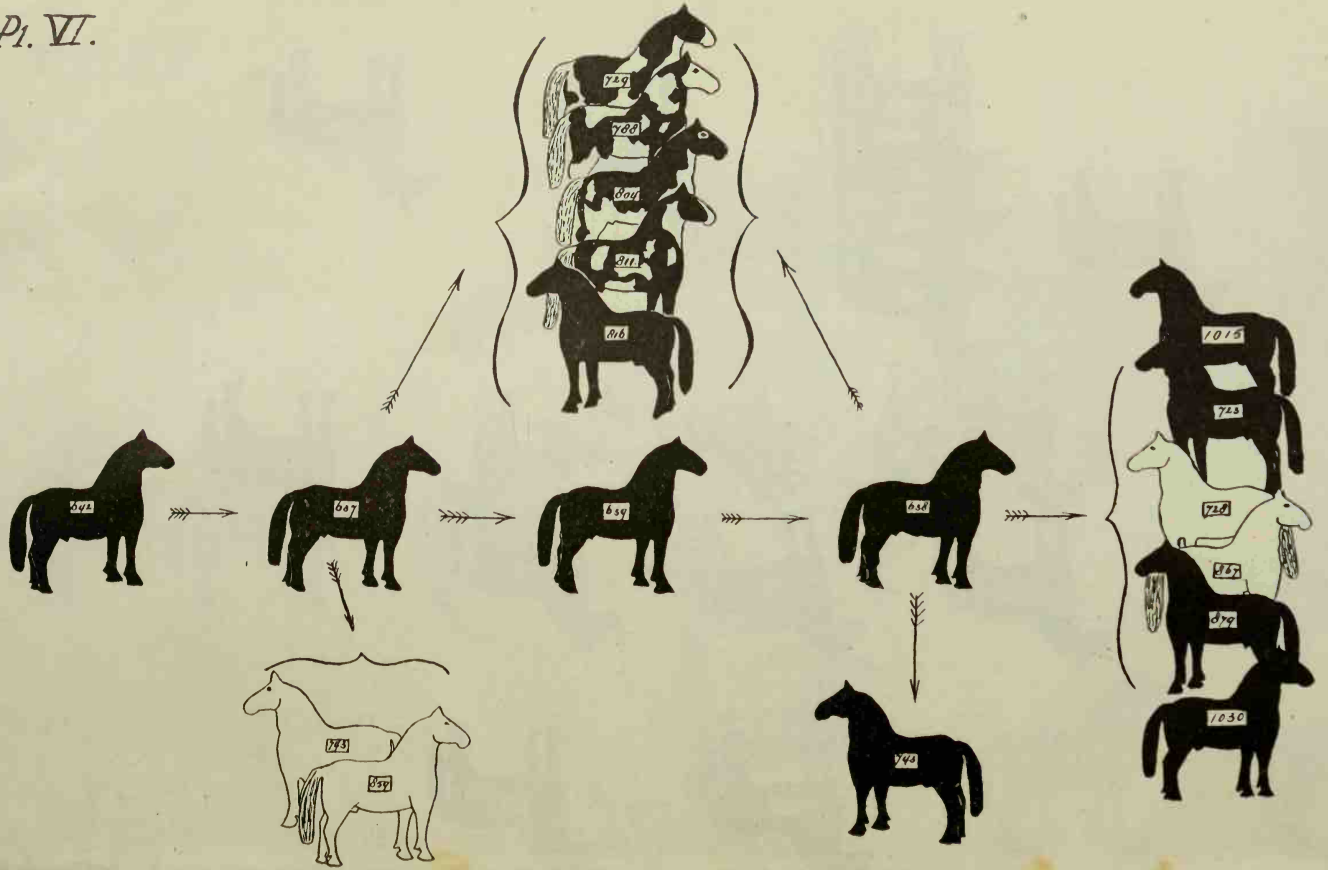
Pl. IV


640
656
656
977
744
855
857
859
867
903
1056
902
873
866
960
939
903
859
967
1032
1033

Pl. V.


562
677
944
934
944
921
953
933
959
1017
1032
938
1022
995
979
969
930
976
920
919

Pl. VI.


729
788
804
811
816
642
647
659
658
744
743
659
1015
743
728
857
877
1030

No. 804. An experimental horse dying six days after an injection of 50 c. c. of blood from No. 638 and 500 c. c. of blood from No. 637.

No. 811. An experimental horse dying three days after an injection of 50 c. c. of blood from No. 638 and 500 c. c. of blood from No. 637.

No. 816. An experimental horse infected by means of an injection with 50 c. c. blood from No. 638 and 500 c. c. of blood from No. 637.

No. 855. Experimental horse infected by means of a subcutaneous injection of 60 c. c. blood serum from No. 636, after having failed to react upon an injection of 100 c. c. of urine from the same source.

No. 856. An experimental horse, giving a doubtful reaction upon a subcutaneous injection of 60 c. c. of blood from No. 857.

No. 857. Experimental horse, infected by means of a subcutaneous injection of 60 c. c. of blood serum from No. 636, after having failed to react upon an injection of 100 c. c. filtered extract of fæces from the same source.

No. 859. Experimental horse, which has failed to react upon a subcutaneous injection of 60 c. c. of blood taken from No. 636, upon a subcutaneous injection of 60 c. c. taken from No. 873, upon a subcutaneous injection of blood taken from No. 637, upon a subcutaneous injection of 60 c. c. of serum from No. 638, and 60 c. c. of serum from No. 637, and upon an intravenous injection of 600 c. c. of blood from No. 919, but which showed an atypic reaction when injected with blood from No. 933.

No. 867. Experimental horse, infected by means of a subcutaneous injection of blood from No. 636, after having failed to react upon a similar injection with the blood of No. 638.

No. 873. An experimental horse, infected by means of a subcutaneous injection of 60 c. c. of blood from No. 855.

No. 879. An experimental horse, infected by means of a subcutaneous injection of 60 c. c. of blood from No. 638.

No. 902. An experimental horse, infected by means of a subcutaneous injection of 120 c. c. of blood from No. 855.

No. 903. Experimental horse infected by a daily dose of 50 c. c. of blood from No. 873, given by the mouth in capsules for ten days. Previous to this, this animal resisted a similar treatment with daily doses of 25 c. c. of blood from No. 636.

No. 919. An experimental horse infected by means of a subcutaneous injection of 120 c. c. of blood from No. 920.

No. 920. A field case from the vicinity of Kelso, N. D.

No. 921. A field case from the vicinity of Shelly, Minn.

No. 924. An experimental horse infected by means of a subcutaneous injection of 120 c. c. of blood of No. 921.

No. 930. An experimental horse infected by means of a subcutaneous injection of 120 c. c. of blood taken from No. 919.

No. 933. An experimental horse infected by means of a subcutaneous injection with 240 c. c. of urine from No. 921.

No. 934. An experimental horse infected by means of giving 2000 c. c. of urine from No. 924 by the mouth.

No. 938. An experimental horse, which failed to react upon a subcutaneous injection with 60 c. c. of blood serum from No. 921.

No. 939. An experimental horse infected by means of a subcutaneous injection of 240 c. c. of blood from No. 873.

No. 944. An experimental horse, infected by means of a subcutaneous injection with 200 c. c. of blood from No. 934.

No. 953. Experimental horse, which resisted an injection of 250 c. c. of blood taken from No. 933.

No. 960. Experimental horse infected by means of a subcutaneous injection of 240 c. c. of blood taken from No. 873.

No. 967. Experimental horse infected by means of a subcutaneous injection of blood from No. 873.

No. 969. Experimental horse infected by means of a subcutaneous injection of blood from No. 919.

No. 976. Experimental horse infected by means of a subcutaneous injection of blood from No. 919.

No. 977. Experimental horse infected by means of a subcutaneous injection of blood from No. 636.

No. 979. Experimental horse infected by means of a subcutaneous injection of blood from No. 919.

No. 995. Experimental horse infected by means of a subcutaneous injection of blood from No. 976.

No. 1015. Experimental horse infected by means of blood from No. 638.

No. 1017. Experimental horse showing a doubtful reaction after an injection with blood of No. 859.

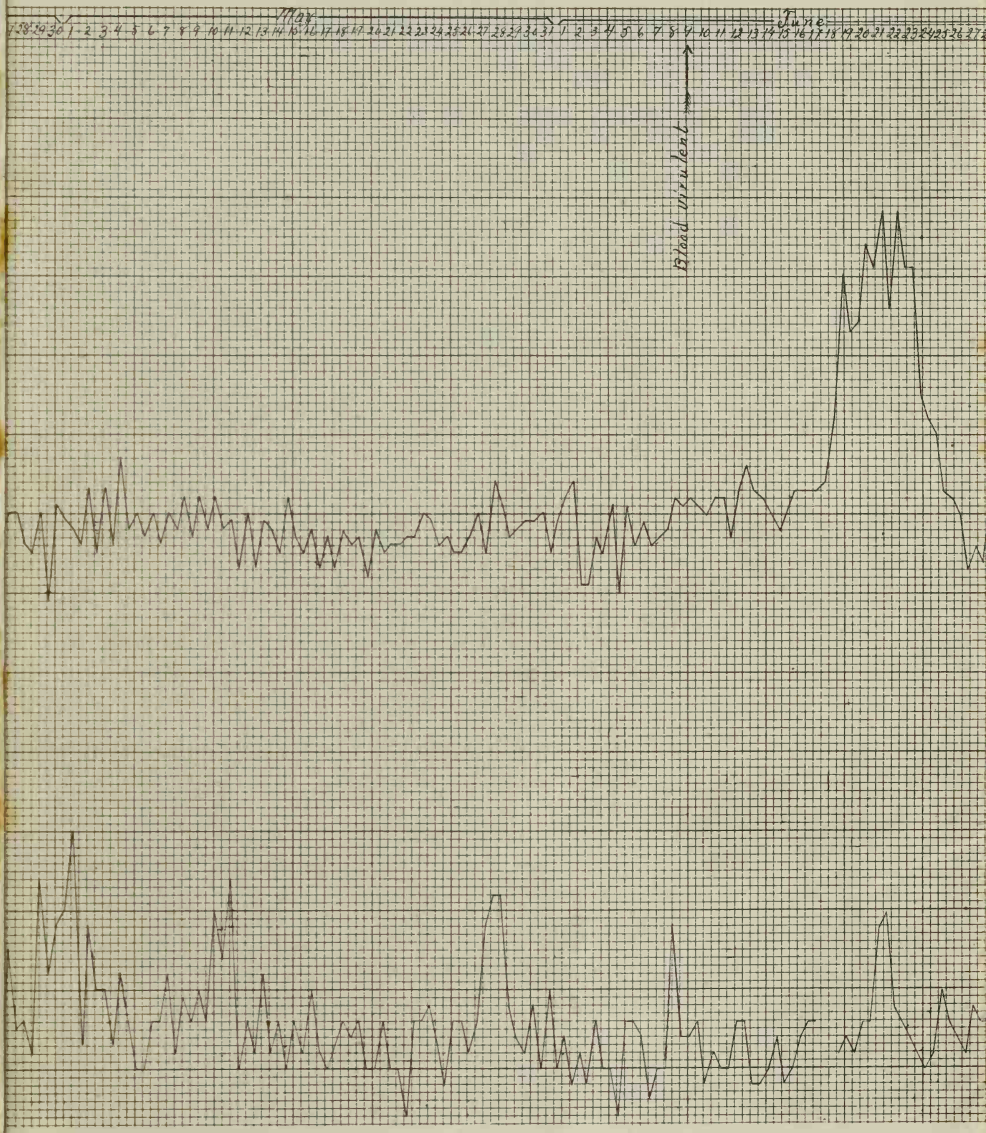
No. 1022. Experimental horse which failed to become infected by the blood of No. 979, but which showed a doubtful reaction after being injected with blood of No. 859.

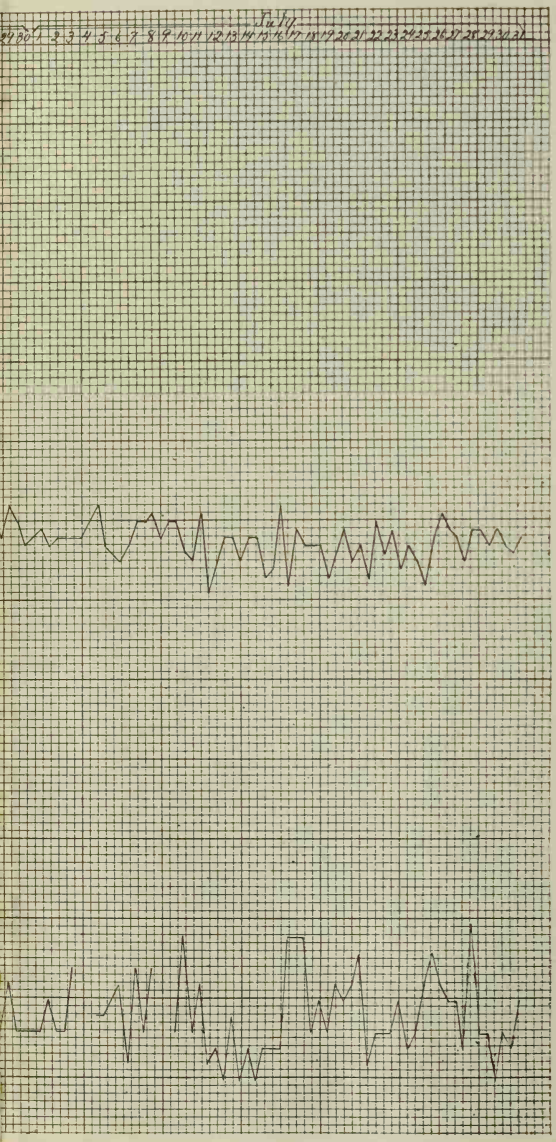
No. 1030. Experimental horse infected by means of blood from No. 638.

No. 1032. A chronic field case obtained from the vicinity of Neche, N. D.

No. 1033. An experimental horse infected by means of the blood of No. 1032.

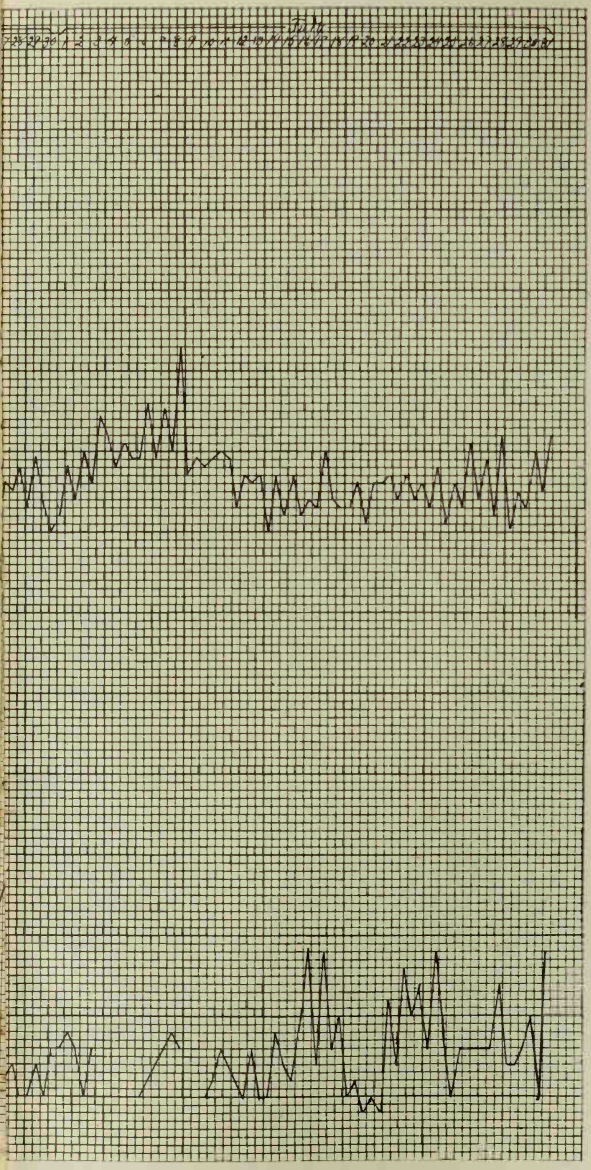
No. 1056. An experimental horse infected by means of the blood of No. 636.

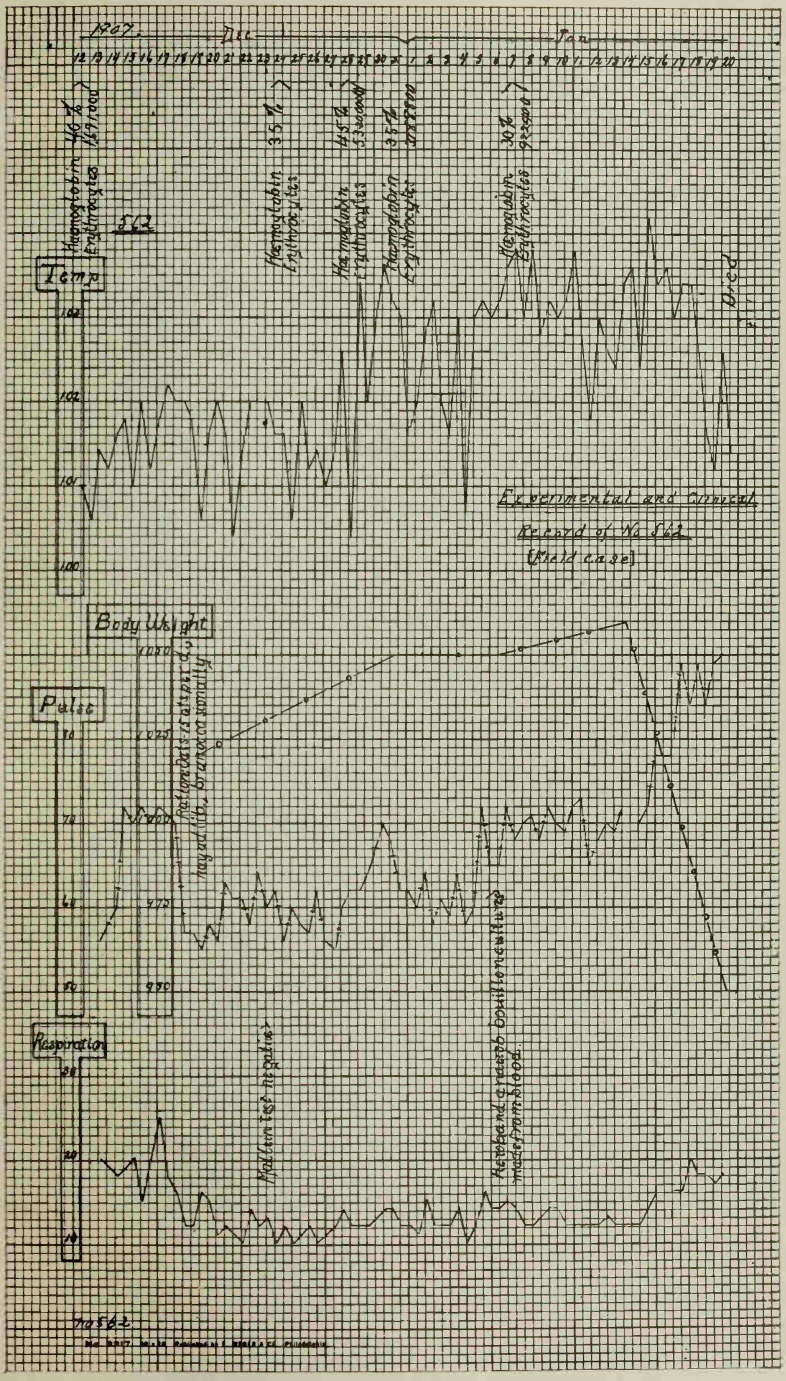

May
June
Blood transfusion


July
29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31

Nov.
Dec.

May
June
Blood pressure




1897.
Dec.
Jan.
Haemoglobin 96%
Erythrocytes 4,671,000
Haemoglobin 35%
Erythrocytes 3,500,000
Haemoglobin 45%
Erythrocytes 5,344,000
Haemoglobin 35%
Erythrocytes 3,500,000
Haemoglobin 30%
Erythrocytes 3,000,000
No. 562
Temp.
Pulse
Body Weight
Respiration
Experimental and Clinical
Record of No. 562.
[Fluid case]
Walton test negative.
Reflexes & nerves Oscillometric readings made per day.
No. 562

1908
Aug
Sept
Experimental and Clinical
Record of No. 440
(Field Case)
Temp
Pulse
Hemoglobin 80%
Hemoglobin 60%
Blood examined
Hemoglobin 50%
Hemoglobin 55%
Hemoglobin 55%
Hemoglobin 55

Sept.

31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7

921

Temp

Pulse

Experimented and Clinical

Record No 921

[Field card]

Blood examined

Blood Virulent

Animal purchased as a field case of Erysipelas standing

Heavy pulse

Many cells possess pleomorphic type

Edema of the scrotum

Increased amount

Edema entirely disappeared

Specific granules of urine low

Reaction acidic pneumococci present

Fatal gradually increased

Reddish urine Hemoglobinuria

Death occurred at 6 P.M. Sept 7th

Shreds of connective tissue

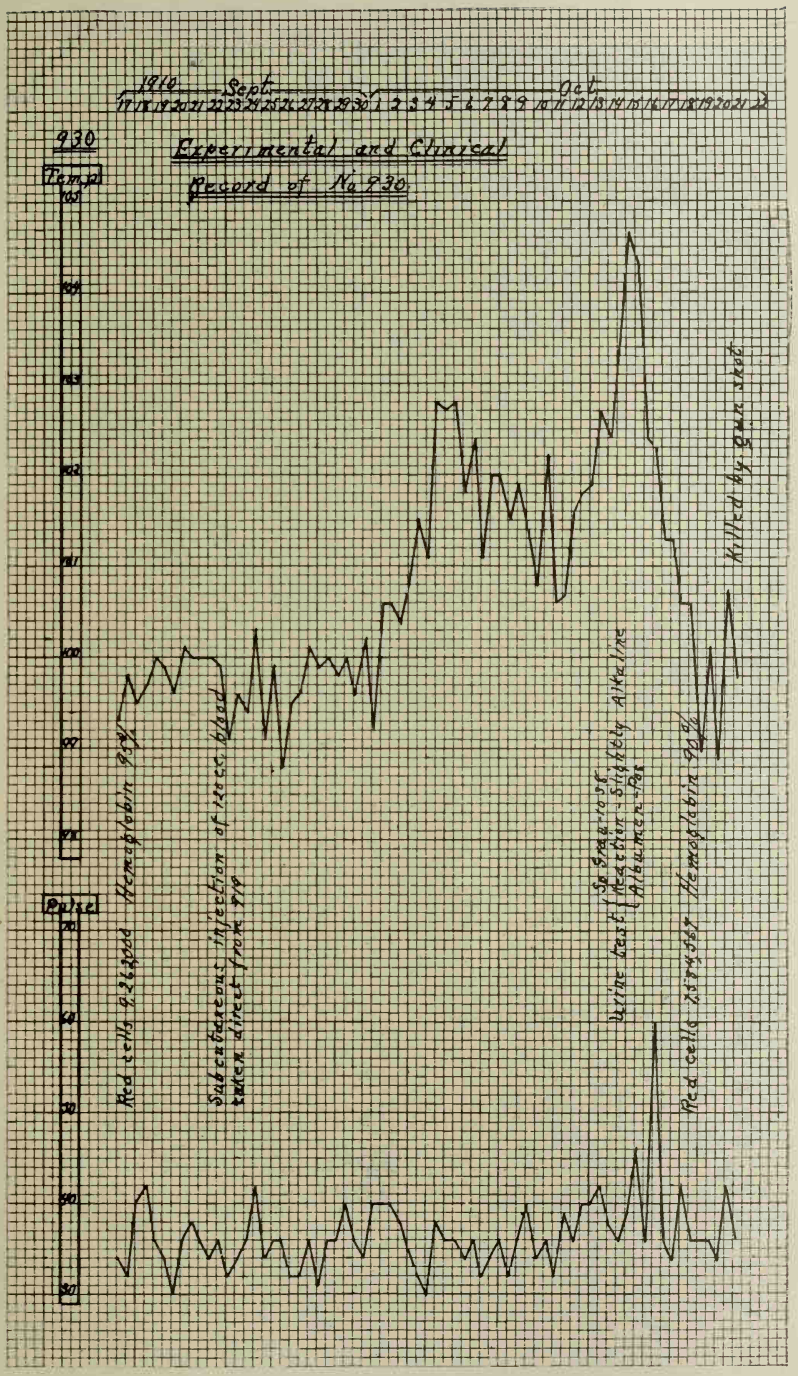
Animal kept under observation for 10 days

Sept.

824 Experimental and Clinical Record of No 824

Temp.

Pulse.


1910
Sept.
Oct.
930
Experimental and Clinical
Record of No 930.
Temp.
Pulse
Red cells 4.26 mill. Hemoglobin 90%
Subcutaneous injection of 100 c.c. blood taken direct from vein
Urine test: Reaction - Slightly Alkaline
Albumen - Nil
Sp. Grav. - 1035
Red cells 5.5 mill. Hemoglobin 95%

1910
Sept.
Oct.
934
Experimental and Clinical
Record of No. 934
Temp.
Pulse

125080